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LV.

THE PREVENTION OF DEAFNESS.*

GEORGE MORRISON COATES, M. D.,

PHILADELPHIA.

The extent to which impairment of hearing has become an economic factor and handicap need scarcely be mentioned to any body of otologists or one interested in public health, and that its prevention, to such degree as may be possible, is also a public health problem that must be studied more than it has in the past, scarcely needs comment here. Evidence that such study is being made, from both the otologic and public health sides, is accumulating, and it is fair to conclude that it is a joint problem that can only be worked out satisfactorily by complete and harmonious co-operation between these two groups.

A review of the known causes of deafness at once suggests certain ways of controlling some of the different types, but the means by which this much to be desired result can be acomplished is another matter. The ideal way of preventing deafness is to take action to control the initial factors that may later cause

*Read by invitation before the Pacific Coast Oto-Ophthalmological Society, May 30, 1931.

impairment of hearing that will eventually progress to such a degree that deafness supervenes. But the prevention of deafness is also concerned with the detection of impaired hearing in its early stages, when normal function can still be restored, or if not that, at least when the condition can be prevented from advancing to the point where deafness is the result. I take it that the term used—deafness—means such impairment of hearing as to seriously handicap the individual in the performance of his or her chief object in life, be it such as school or college education, the pursuit of gainful or altruistic occupations, or taking one's proper place in society and in the life of the home. Thus the study of preventive measures falls roughly into two classes: measures directed towards eliminating the known underlying causes of deafness, and on the other hand, diagnostic and therapeutic measures for detecting and curing the malady at a time when cure is still possible, or of preventing its otherwise inevitable progression.

What are the underlying causes that can be dealt with under the first head? Some of them are: Syphilis, the acute contagious diseases and infections, hypertrophied and infected tonsils and adenoids, all infections of the upper respiratory passages and possibly some abnormalities of the nasal passages, the marriage of those persons having a family history of deafmutism or who are congenitally deaf, or of those with a family history suggesting otosclerosis, the curtailment of industrial and other noises, or the minimizing of exposure thereto, care in the exposure to noises caused by firearms, either at the target or in warfare, the proper surgical treatment of suppurations of the middle ear and its adnexa, and in the management of brain tumors, the use or administration of certain detrimental drugs, possibly including tobacco and alcohol, proper care of the nose and ears in bathing and swimming.

In the prevention of deafness by means of eliminating, as far as possible, the underlying causes, various agencies must be utilized and harmonious co-operation should obtain among those directing them, as the problem is a vast one and can only hope for some measure of success after long continued effort. These

agencies include: (1) Research, necessarily heavily endowed, such as is now being conducted by the American Otological Society, into the underlying causes of various forms of progressive deafness, the etiology and pathology of development of which are now obscure; (2) education, both of the medical profession, which still needs it, the public health authorities aside from the medical profession, the managers of certain industries which imperil the hearing of their workers, the school authorities and the laity; (3) propaganda to enforce this education, (4) legislation to help secure the desired results, and (5) the awaking of the minds of the mass of the people to the great economic waste resulting from what appears to be an increase of impaired hearing. There are few jobs where a worker with impaired hearing is not handicapped or where his efficiency is not in some degree diminished, and some occupations that are absolutely barred to one with defective hearing. When we consider the often quoted estimate of three million public school children with a loss of hearing in many definite enough to retard their education and to cause them to repeat one or more grades, the cost to the taxpayers becomes at once apparent, and when to this is added the deafened school children of other years who are now workers under the handicap of deafness, as well as those of pre-school age with impaired hearing, the total becomes staggering and calls for some concentrated effort to obtain a partial solution. That a complete solution will ever result is most improbable.

To take up in a little more detail the elimination of the causative factors, we can see some possible ways for at least making a start towards the solution of this problem. Syphilis is a problem for public health authorities and preventive medicine. Education will help here, moral training should not be ignored; any means that will minimize the innocent acquisition of the disease; education in venereal prophylaxis, as practiced in the army and the navy; quarantining during the infectious stage, perhaps, and this must necessarily make the reporting of such cases obligatory by proper legislation. The discovery of still more efficient methods of curing syphilis before the nervous system is attacked should also result in lessening the cases of syphilitic neurolabyrinthitis with the accompanying deafness.

Beyond question the control of the acute exanthemata, now well under way and giving promise of much greater results in the future, will eliminate many ear infections, now commonly seen in measles and scarlet fever especially, thus preventing both the deafness from immediate damage to the middle ear, the occasional labyrinth involvements, as well as the progressive deafness of young adult life that often has its origin in such middle ear infections in youth, even when the infection runs an apparently mild course. When epidemic cerebrospinal meningitis is controlled, deafness from that source will be automatically eliminated also, and epidemic parotiditis comes in the same class. The specific cause of the acute cold and influenza is not determined, though some progress has been made towards preventing the spread of these diseases. More research is needed here, but isolation of infective persons, the avoidance of promiscuous sneezing and coughing in public places, the barring of infected children from schools, as now practiced, will all help. Building up the resistance of susceptible individuals, whether by specific immunization or by measures of general hygiene and sanitation, dieting and the improvement of living standards will contribute to this end.

We must consider the much mooted question of the operation of tonsillo-adenoidectomy in the light of its possible prophylactic value in the prevention of impaired hearing or deafness, and here it is difficult to draw a hard and fast line that will be acceptable to all. Though some may dispute the harm that hypertrophied, or more properly, diseased tonsils cause the hearing apparatus, few will deny that the presence of an adenoid mass of any considerable size, in the nasopharynx, and more especially lateral masses in the fossæ of Rosenmüller, encroaching on the lip of the eustachian tube, is a serious menace to hearing. It, therefore, seems the part of wisdom to destroy such a potential cause of deafness at an early age, before, and not after any damage is done. Whether tonsils that are as yet only suspicious should be removed at the same time is a controversial subject, but it seems to the writer that such is the best course of action, and gross abnormalities of the septum seriously affecting normal ventilation and breathing, or that are conducive to the establishment

of sinus infections, should receive attention, as well as the often overlooked infection of the accessory sinuses of children. If we believe, with Love, that in the intermarriage of the congenitally deaf the offspring will follow the Mendelian law and be deaf also, some way must be found to prohibit such marriages, though that is difficult because, as that writer points out, the deaf are often thrown together by force of circumstances, so that attachment and marriage follow naturally. It must not be forgotten that this problem is still more complicated by the fact that congenitally deaf children may spring from the union of those who, having normal hearing themselves, yet carry a family taint of deafness of this character. Obviously those suffering from acquired deafness do not fall within this category, but the otosclerotic does. And yet who can say with certainty that a family history showing one or more deafened in the two preceding generations is necessarily that of otosclerosis? When consulted for advice as to the result of marriage under the circumstances, this question is difficult to decide.

Industrial noises, as well as those of warfare, either when continuous, comparatively small noises, as those caused by riveters —the so-called boilermaker's deafness—or the single concussion of a high explosive in battle, cause definite and permanent destruction of part or all of Corti's organ. The remedy is a problem for industrial medicine to solve. The obvious way is so to muffle the noise of the machine shop or construction worker that harm will not result, but this cannot be accomplished in warfare. The alternative lies in providing a muffler for the individual ears exposed. These ear protectors, of which there are many designs, worked well enough during the late war, when the soldiers could be persuaded to use them or when not taken by surprise. For the most part they were not used at all, partly because the hearing function was needed for learning commands, directions or warnings that, being neglected, might well result in loss of life. It must not be forgotten that exposure to the noise of artillery or small-arms practice may be as equally destructive to the hearing function as actual battle noises. What part the ordinary noises of our city streets play in the production of deafness is an open question. Of these, construction noises and those of modern

transportation would seem to be the worst, but whether they are intense or constant enough to cause serious damage must be determined by further study. It is probable that they have some significance, if only, as suggested by Wells, by causing a general high nervous tension or neurasthenia by their irritating quality and by interference with normal sleep and rest. It may be that the organ of hearing will in time adjust itself to such disturbances, or as a result of them the hearing of city dwellers will eventually decline. The remedy may lie between education, better mechanical construction of automobiles and trolley cars, quieter engineering methods and restrictive legislation. The latter has been tried and so far has dismally failed. Neither has prohibition perceptibly diminished the vocal noises in the city streets that still interfere with normal rest.

The avoidance of certain drugs, the arsenicals, salicylates and quinin, for instance, is purely a matter of medical regulation. In this classification also must be considered alcohol and tobacco. Neither of the latter, taken temperately, is detrimental, but in excess, and in the case of alcohol, when impure, damage can be caused the perceptive apparatus, or existing damage to the conductive mechanism may be increased. Either of these drugs, used intemperately, as they more frequently are nowadays, may hasten the course of an otosclerosis. Legislation has not and will not solve this problem—the true solution lies in early training in habits of temperance and self control, for prohibition, while it does many things, does not prohibit.

The proper and skillful treatment of the nose and throat of those suffering from the acute contagious diseases will prevent many cases of middle ear infection and subsequent deafness, and if the middle ear becomes infected skillful attention, both during and after the attack, will prevent or minimize the damage. As a general rule it can be stated that the shorter the duration of the ear discharge the less permanent damage will result. If the mastoid is involved, operation, not too early before barriers of resistance have been established, nor yet with too long a delay, allowing irretrievable damage to the middle ear structures, will give the best results. The mastoid operation should be most complete and

thorough, embracing the removal of all accessible cells, whether infected or not, even in some cases involving the search for the pneumatic spaces of the petrous pyramid, as urged by Profant, Kopetzky and Almour. Overlooked infected cells cause prolonged suppuration, slow healing, continuing middle ear discharge and the resultant hearing loss. The most commonly overlooked cells lie in the posterior root of the zygoma, the deep layer of cells in and behind the posterior wall of the external auditory canal, in front of the sigmoid sinus, and those over and also just below the knee of the sinus. The tip is, as a rule, well cleaned out.

Middle ear infections, as well as infections of the accessory nasal sinuses, which may in turn cause loss of hearing, either by causing ear suppuration or by acting as a focus for the later production of perceptive deafness, are frequently caused by bathing and swimming, and are due particularly to the modern style of swimming with the face under water, deep and fancy diving, and to a lesser degree to infected water in public bathing pools. The latter can be controlled by adequate municipal or other inspection and examination, with appropriate regulation, but the style of swimming can hardly be changed in this age, which demands speed above all things. Proper medical instruction in regard to nose blowing will minimize the danger, while the use of ear plugs or surf protectors will obviate many recurrences in latent cases of chronic middle ear suppuration with patent perforations.

Detection of incipient losses of hearing is of the utmost importance if restoration to normal is to be obtained, or if progression to an incapacitating degree of deafness is to be avoided. The various agencies at our disposal are many, and it is distinctly a combined work for the otologists and the public health and school authorities. Much progress has resulted, and more will ultimately result as facilities become available, from the aural examination of school children. Older methods have been superseded by the employment of the 4-A audiometer by means of which forty or more children of school age can be tested simultaneously by a teacher or others who have had a little instruction. This method is rapid and gives a rough classification of the nor-

mal, the doubtful cases, and those who are indubitably defective. In the latter class a more extensive diagnostic examination is essential, and these children must go, for individual examination, to a private otologist or to a "prevention of deafness" clinic. Such clinics are now being established in many hospitals and medical teaching institutions. Their function is to determine the type and degree of the hearing impairment, to suggest proper treatment and to refer the case, through the social service, to the appropriate department where such treatment can be carried out. To be most effective, such a clinic must be separated from the general otologic department where the required time and attention would not be likely to be given; its function is diagnostic and advisory only, the examiner thus being enabled to give his individual attention to his very specialized subject, and the work of such a clinic is, of course, not limited to cases sent from the schools. It includes the child of preschool age, often a very difficult case to handle, as well as those who have reached adult life. Here perceptive deafness can be diagnosed as possibly due to intracranial neoplasm and the case referred to the neurosurgeon, cases of chronic suppurative otitis media sent in for intratympanic or radical surgery, foci of infection in the throat, nasopharynx or sinuses diagnosed and sent to the appropriate department. Aural examinations of employees of industrial establishments are usually rough and ready but sufficiently effective to detect any definite defect. Such cases are also sent to the prevention or diagnostic clinic for more careful study and advice. Funds are needed to further this work in the public and other schools, as well as education of school authorities as to its need and value. Funds are also needed by the hospitals for the establishment of many more diagnostic clinics. To their credit be it said, aurists can usually be found to donate their services to this work if facilities can be provided for them to work with.

Checking of impaired hearing, when detected, must depend upon thorough study of the individual case, a very careful history and the elimination of all causative factors discoverable. Aside from this, remedial measures include the prompt checking of acute aural discharge when possible, and more particularly, after-care consisting of gentle inflation and massage, which is often omitted.

In chronic suppurations appropriate surgery, designed to stop the discharge and its accompanying destruction, and to remove obstructions to the reception of sound impulses, will often restore a fair amount of hearing acuity and prevent further loss. The different types of tympanic and mastoid surgery suit various pathologic conditions and various degrees of residual hearing. When the middle ear is principally at fault, proper drainage obtained by enlargement of an existing perforation, the removal of obstructing granulation and polypi, attic drainage, as suggested by Babbitt, or ossiculectomy, as modified by Tobey, all, if successfully performed, may result in improved hearing conditions. The modified radical operation, of which there are various types, preserves hearing when the middle ear is still a usefully functioning organ, and recent studies by Ersner and Pressman indicate that even the radical mastoid operation, when properly done, may be counted upon to improve hearing or at least preserve what remains.

The prevention of deafness clinic must, therefore, work in close co-operation with a properly conducted treatment and operative service designed to continue the initial study and carry out the recommendations of the group working on the detection and diagnosis of hearing impairment. Social service co-operation is of the utmost importance in this work. Its functions include the contact with the school examining authorities, the bringing in of the doubtful cases for more careful study, the follow up of the cases under study to ensure proper treatment, bringing cases back at intervals for checking results, finding proper situations for those suffering from occupational forms of deafness where it is imperative that harmful positions be abandoned, and the keeping of statistics and records.

The vastness of the program outlined is appalling, and it is very evident that time, money, energy and a host of conscientious workers are all necessary in order to obtain even partial results. That any panacea will be found is impossible because of the multitude of etiologic factors to be contended with, and there is no substitute for hard work by all interested. Much stimulation and help has been and is being obtained from the deafened them-

selves through their local leagues for the hard of hearing, speech-reading clubs or similar organizations, and the American Federation of Organizations for the Hard of Hearing. All of these societies deserve the active help and support of every otologist, for it is only by concerted effort that any great good can be accomplished.

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LVI.

SOME ASPECTS OF CERTAIN OF THE CRANIAL NERVES.*

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An understanding of the complex anatomic relationships of some of the nerves of the head region in man involves a knowledge of their phylogenetic history. Comparative anatomists have given us the principal facts of this history in the vertebrate series. We may gain a foundation for later considerations by passing briefly in review the more important aspects of this accumulated knowledge with especial reference to some of the nerves with which the otolaryngologist comes into most intimate contact.

The trigeminus nerve in the fishes has four branches, namely, the superficial ophthalmic, the deep ophthalmic, the maxillary and the mandibular. The deep ophthalmic branch has its own ganglion and appears originally to have been the sensory part of the primitive nerve whose motor portion becomes the trochlearis. In the land vertebrates the deep ophthalmic nerve appears to have fused with the superficial ophthalmic, forming the ophthalmic division of the trigeminus of human anatomy. The primitive relations of the deep ophthalmic nerve to the fourth nerve, for which comparative anatomists have some evidence, is completely lost in adult mammals and man. The superficial ophthalmic and the maxillary branches of the trigeminus are purely somatic sensory, supplying the skin of the head and part of the mouth membrane. The mandibular branch contains a motor component in addition to somatic sensory fibers.

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*Read before the Western Section of the American Laryngological, Rhinological and Otological Society, Seattle, January 31, 1931.

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It will be noted that there is a rostrally directed branch, the ophthalmic; another branch, the maxillary, passes in front of the mouth cleft; and a third branch, the mandibular, which passes behind the mouth cleft. Many comparative anatomists regard the mouth as a modified gill cleft, and the maxillary and mandibular nerves, accordingly, as pre-trematic and post-trematic branches, respectively, of the trigeminus nerve, which supplies this head segment.

In considering the facial nerve we find a similar triple branching, forming the palatine branch of the seventh, the internal mandibular branch and the hyomandibular branch. The palatine branch contains sensory fibers which reach the mucous membrane of the pharynx rather than an external surface of the body. These fibers are therefore visceral sensory rather than somatic sensory. The internal mandibular seventh branch also contains special sensory fibers to the taste buds.

In the water living forms the facialis has three special branches which supply the so-called lateral-line organs of the head region. These branches, the superficial ophthalmic seventh, the buccal seventh and the lateral-line part of the external mandibular branch of the facial nerve disappear completely with the assumption of the land habit.

The palatine seventh remains without much modification save some relative reduction in size, and corresponds to the superficial ophthalmic fifth nerve. The internal mandibular nerve passes in front of the opening of the spiracle and is to be regarded as the pre-trematic branch of the seventh, although some anatomists consider it as the post-trematic branch. In its peripheral course this nerve passes along the articulation between the quadrate bone and the condyle of the jaw. In the mammals, in which these two bones have been drawn up into the middle ear in the further process of the development of the head and have become modified into the incus and the malleus, respectively, this nerve, which we now recognize as the chorda tympani, retains its primitive relations, passing between the ear ossicles, although the course of the nerve itself has been modified in the process. These facts of morphologic development explain some of the peculiar features

of the anatomy of the middle ear. Peripherally the chorda tympani continues as the lingual branch of the seventh, but since it supplies the same region as does the lingual fifth nerve the two become united in one sheath to form the lingual nerve of human anatomy.

The hyomandibular seventh nerve in water forms supplies motor fibers to the lower jaw and to the hyoid gill arch. When this muscle mass is reduced in the lower land-living forms the motor hyomandibular seventh becomes also greatly reduced in size. In mammals, however, the platysma muscle and the mimetic muscles which are derived as slips from the platysma, are developed from that part of the branchial musculature which is innervated by the facial nerve. These muscles retain the primitive innervation, and as they become more and more differentiated and grow in size, the motor element of the facialis again increases in importance, so that in mammals it constitutes the greater part of the seventh nerve.

In some of the lower vertebrates there has been described a cutaneous branch of the seventh nerve, supplying the surface of the hyoid region. This branch has also been found in the mouse by Rhinehart,[8] and was reported in the human embryo by Larsell and Fenton.[5] We may recognize then in man the same fundamental features of structure and even many of the details in the cranial nerves, which are present in the less modified head region of lower vertebrates.

With the development of the sympathetic system in the head region the palatine branch of the seventh nerve becomes modified from its primitive condition of an apparently simple visceral sensory nerve into a mixed nerve. There is formed upon it a sympathetic ganglion, the sphenopalatine, which is composed entirely of multipolar sympathetic ganglion cells. Leading to the ganglion and taking their course with the palatine seventh fibers we have found numerous fine myelinated fibers, which histologically and in other respects must be regarded as preganglionic fibers which have their origin in the bulb and their terminal connections in the sphenopalatine ganglion. Together with the palatine seventh fibers they constitute the great superficial petrosal nerve of hu-

man anatomy. The sensory or palatine seventh fibers are not interrupted in the sphenopalatine ganglion, but merely pass through it and join with branches of the maxillary nerve to form the palatine and posterior nasal nerves.

Kuré and Sakurasawa[4] have recently made an experimental study of the sphenopalatine ganglion and find that it receives preganglionic fibers from the maxillary nerve as well as from the great superficial petrosal. These are the small myelinated fibers contained in the nerves named. Together with the postganglionic fibers which arise from cells in the sphenopalatine ganglion, they belong to the parasympathetic or cranial autonomic system of nerves. Kuré and Sakurasawa have experimental evidence that the unmyelinated fibers in both the vidian and the maxillary nerves completely disappear after extirpation of the superior cervical ganglion and hence are sympathetic or thoracicolumbar autonomic fibers. They pass through the sphenopalatine ganglion without interruption, as has previously been stated, on anatomic grounds (Müller,[6] Larsell and Fenton[5]). This statement is now confirmed experimentally in the work cited.

We find a bundle of unmyelinated fibers from the deep petrosal nerve, the internal carotid plexus and the superior cervical ganglion, which passes through the sphenopalatine ganglion and into the zygomaticotemporal nerve. These are undoubtedly postganglionic fibers from the superior cervical ganglion to the lacrimal gland, but the evidence is not clear whether they are vasomotor or direct secretory fibers. The sphenopalatine ganglion itself sends postganglionic fibers to the tear gland through the zygomaticotemporal nerve and the lacrimal branch of the ophthalmic nerve, according to Müller.[6] It also supplies secretory fibers to the mucous glands of the nasal cavity. Kuré and Sakuarasawa believe that these preganglionic fibers come to the ganglion through the maxillary roots as well as through the great superficial petrosal nerve, since secretion continues after section of the vidian nerve and the chorda tympani but ceases completely after extirpation of the sphenopalatine ganglion, whose only remaining connection with the central nervous system is through the maxillary nerve.

The sensory fibers of the great superficial petrosal nerve have their ganglion cells, not in the sphenopalatine ganglion, which is purely autonomic, as stated above, but in the geniculate ganglion. Here also, we have reason to believe, are located the ganglion cells of the cutaneous branch of the facialis which is distributed to the tympanum, the back of the external ear and the mastoid region. In this relationship we have a possible mechanism, comparable to that elsewhere in the body, for reference of pain from the sphenopalatine region to the ear region, as has been described elsewhere.

If we analyze the glossopharyngeal nerve from the same morphologic point of view, we find again an anterior branch, together with a pre-trematic and a post-trematic branch, in relation to the first gill slit. The palatine ninth nerve corresponds in part to the nerve of Jacobson. In our series of Pal-Weigert sections of this region, made from adult human material, we find myelinated fibers of large and medium size which appear to have their cells of origin in the petrous ganglion and which pass through the nerve of Jacobson into the superior and inferior caroticotympanic nerves (the small deep petrosal nerve of some authors). The small superficial petrosal nerve, to which Jacobson's nerve also gives rise, carries only small myelinated and unmyelinated fibers to the otic ganglion. The superior caroticotympanic nerve sometimes joins with the great superficial petrosal, according to Spalteholz. In one of our series of Pal-Weigert preparations we have been able to follow large myelinated fibers from the nerve of Jacobson into the superior caroticotympanic nerve and forward to the wall of the internal carotid artery (Fig. 1). Here the fibers were lost in this series, but they did not appear to become part of the carotid plexus, and had probably been severed in the difficult dissection necessary before the mass of nerves could be prepared for microscopic sections. In another series a small bundle of myelinated fibers was followed from the vidian nerve, where it is included in a common sheath with the great superficial petrosal part of the vidian nerve, into the great superficial petrosal nerve where the latter is distinct from the great deep petrosal, and finally along the internal carotid artery for some distance as a bundle quite distinct from the great super-

ficial petrosal. Selected sections from this series are illustrated in Figs. 2, 3 and 4. The series unfortunately did not include the nerve of Jacobson and the tympanic plexus, but there appears little doubt that the bundle in question is the superior caroticotympanic nerve. The large and medium size myelin fibers from the petrous ganglion, passing through the nerve of Jacobson, the superior caroticotympanic nerve and the great superficial petrosal nerve to the sphenopalatine ganglion would constitute the third sensory root of this ganglion referred to by Schäfer and Symington (Quain's Anatomy, vol. III, pt. II, p. 22). These fibers, however, like those from the geniculate ganglion, must be regarded as passing through the sphenopalatine ganglion and into nerves of distribution to the palate. They would correspond to the palatine ninth branch of lower vertebrates. The ninth nerve, like the seventh, also gives rise to a lingual branch which, with the phylogenetic development of the tongue, becomes prominent. The glossopharyngeus also has a cutaneous branch which joins with the auricularis vagi and the cutaneous seventh to supply some of the skin of the external ear and adjacent regions. There is also a connecting branch between the petrous ganglion and the superior cervical ganglion which contains some myelinated fibers of medium size. These, however, disappear in the capsule of the ganglion.

The vagus includes the nerves which supply four or five of the branchial segments. In lower forms there are pre-trematic and post-trematic branches. In higher forms, with absence of gill clefts in the adult, there is a marked modification of the nerve, but it still has its origin from the bulb by an extensive series of rootlets. Phylogenetically and embryologically the bulbar part of the spinal accessory nerve must also be included with the vagus nerve. The rootlets which give rise to the spinal accessory nerve have dorsal ganglia in the embryo (Streeter,[10] but these ganglion cells usually are regarded as disappearing entirely, or as is likely, are all drawn into the root (jugular) ganglion of the vagus in further development. Not infrequently traces of the spinal accessory ganglia persist in the adult, according to Schäfer and Symington.[9] The result is that the spinal accessory nerve of the adult is regarded as purely motor, supplying

two muscles, the trapezius and sterno-cleido-mastoideus, which are derived from the branchial musculature.[1]

There is a connecting branch between the vagus ganglia and the glossopharyngeal ganglia in which we have found large myelinated fibers in rabbit and human material. Whether these fibers arise in the jugular ganglion or in the petrous ganglion is not known. There are also connecting branches between the nodose ganglion of the vagus and the superior cervical ganglion, but these contain only unmyelinated and small myelinated fibers.

We have found in our sections a bundle of myelinated fibers in the internal carotid plexus, which enters the superior cervical ganglion with the unmyelinated fibers and extends through the main mass of the ganglion. In the opposite direction these fibers have their course with the internal carotid plexus to the great deep petrosal nerve, with which they then continue to the sphenopalatine ganglion. As to their origin, we can only say that they do not appear to arise in the superior cervical ganglion. Dr. Kuntz informs us, in a personal communication, that he has removed the upper thoracic root ganglia in the cat without effect on a similar bundle.

From the preceding anatomic description it is evident that the crowded region of the sphenopalatine ganglion is a cross-roads through which diverse fibers take their course. There are (1) sensory fibers of the palatine seventh nerve from the geniculate ganglion and apparently sensory fibers from the petrous ganglion of the glossopharyngeus; (2) there are cranial autonomic secretory fibers from the great superficial petrosal nerve and the maxillary nerve; (3) probable thoracicolumbar secretory fibers from the superior cervical ganglion; (4) vasoconstrictor fibers from the superior cervical ganglion; (5) vasodilator fibers from the

1. Since this paper has gone to press there has appeared an abstract by William F. Windle (Wistar Institute Bibliographic Service, advance abstract sheet, May 5, 1931) in which it is stated that the spinal portion of the spinal accessory nerve in the cat and monkey is a mixed nerve, with a few nerve cells of sensory type, and sometimes small ganglia, located along its course. Section of the nerve where it enters the sternocleidomastoid muscle, according to Windle, produces chromatolytic changes in most of the nerve cells mentioned. He considers these cells and fibers as probably proprioceptive in function.

great superficial petrosal and maxillary nerves; and (6) fibers of sensory type which pass through the superior cervical ganglion but whose origin is unknown.

These anatomic facts suggest some possibilities of explaining certain clinical phenomena which require further investigation by experimental methods which cannot be applied to human material. Among these may be named the question already raised as to the origin of the bundle of myelinated fibers common to the great deep petrosal and internal carotid nerves. Also, what is the fate of the ganglion cells found in the embryo on the roots of the spinal accessory nerve? Are they the ganglion cells of muscle sense fibers which pass to the trapezius and sternomastoid muscles with the motor roots of the accessory nerve? If so, they would correspond to ganglion cells found by Nicholson[7] along the oculomotor nerve and by Allen[1] along the motor root of the trigeminus. The further question is then raised as to the relation of such possible muscle sense fibers of the accessory nerve to the myalgic pains sometimes occurring in the neck region when the sphenopolatine ganglion region is involved by infection or otherwise. These muscles, the sternomastoid in particular, as histologic preparations show, are rich in neuromuscular spindles of the type considered as serving muscle sense. The anatomic connections do not, however, show any peripheral relations of nerves or ganglia which would offer an adequate explanation for reference of pain. There are possibilities in the central connections which require further study. There are also possibilities associated with the vasomotor centers and their connections which are intriguing but concerning which we know very little. There is here a field for experimental and clinical studies which should yield interesting results.

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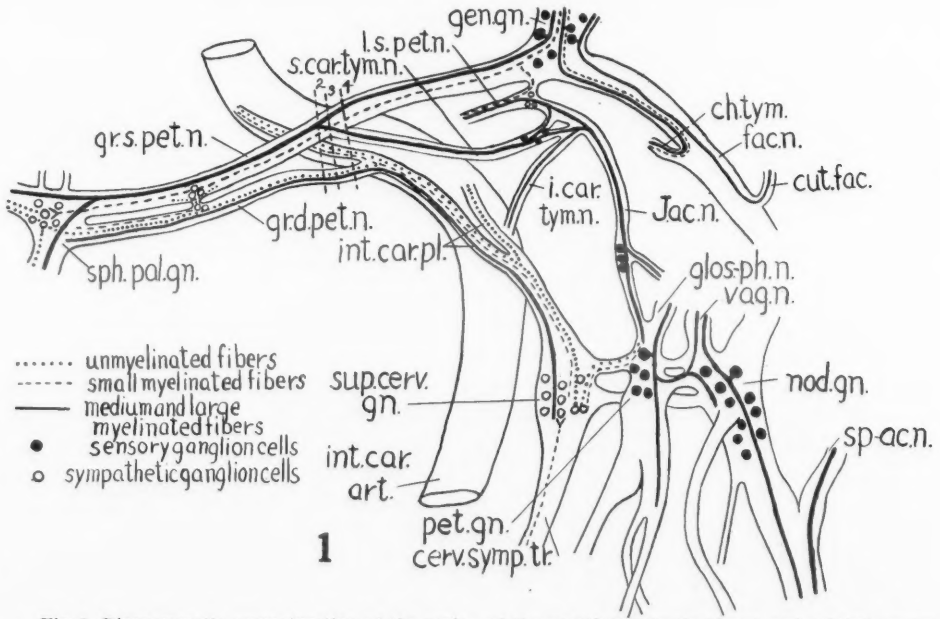
ABBREVIATIONS FOR ALL FIGURES.

ch. tym.—Chorda tympani.
cerv. symp. tr.—Cervical sympathetic trunk.
cut. fac.—Cutaneous branch of facial nerve.
gen. gn.—Geniculate ganglion.
fac. n.—Facial nerve.
glos. ph. n.—Glossopharyngeal nerve.
gr. d. pet n.—Great deep petrosal nerve.
gr. s. pet. n.—Great superficial petrosal nerve.
int. car. art.—Internal carotid artery.
int. car. pl.—Internal carotid plexus.
i. car. tym. n.—Inferior carotico-tympanic nerve.
Jac. n.—Nerve of Jacobson.
l. s. pet. n.—Lesser superficial petrosal nerve.
nod. gn.—nodose ganglion.
pet. gn.—Petrous ganglion.
sp. ac. n.—Spinal accessory nerve.
sph. pal. gn.—sphenopalatine ganglion.
s. car. tym. n.—Superior carotico-tympanic nerve.
sup. cerv. gn.—Superior cervical ganglion.
unmy b.—unmyelinated bundles.
vag. n.—Vagus nerve.



UNIV. OF MICH.

Fig. 1. Diagrammatic reconstruction of the region of the caroticotympanic plexus and related nerves in man showing the paths of the different histological types of nerve fibers. The various nerves shown are represented out of proportion as to size and in somewhat distorted relationships for the sake of clearness in indicating fiber paths.

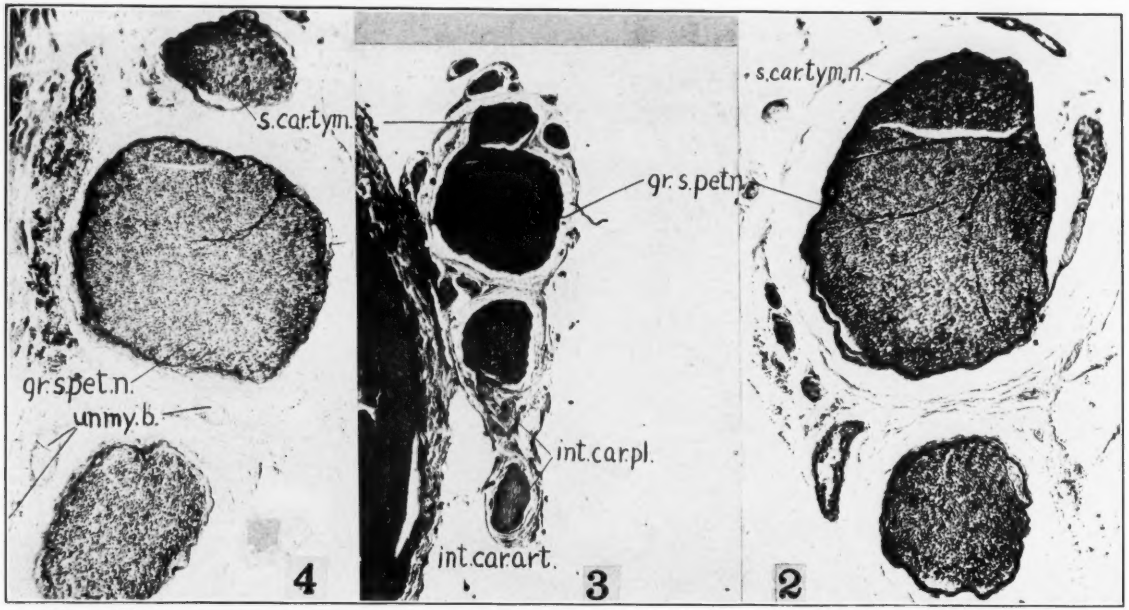


Fig. 2. The great superficial petrosal and related nerves at level indicated by 2 in Fig. 1. Photomicrograph. Pal-Weigert stai. Fig. 3. Same nerves at level 3 in Fig. 1. Fig. 4. Same nerves at level 4 of Fig. 1.

LVII.

PAROTID FISTULÆ IN THE MASTOID WOUND, WITH REPORT OF CASE.*

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Philadelphia.

A review of the literature pertaining to parotid fistulæ following the mastoid operation has shown such a remarkable coincidence in record that it would seem more appropriate to discuss them as a group, and, with the permission of the society, the title of this brief paper will be so modified. The case to be described here has not proven unique at all, but is one of a comparatively small number, perhaps twenty-five in all, whose onset, history and treatment have had much in common. Grove,[1] in 1923, compiled a list of fifteen cases reported prior to his own, the first to be recorded with bilateral fistula following the mastoid operation. To his list may now be added single cases of Davis,[2] Boot,[3] Trostler[4] and Simpson,[5] and two of Kausch.[6]

It is rather unusual to present conclusions at the outset of a paper, but at least three of the incidents pertaining to parotid fistulæ in the mastoid wound are so striking that they might well be adopted as themes in discussion. First, the occurrence of parotid fistula seems scarcely justified by the mastoid operative procedure alone; second, in view of the important normal and abnormal relations to the operative field, the number of fistula accidents is remarkably small; third, the efforts at repair have been varied and doubtless skillful but often unsuccessful.

The occurrence of the fistula has apparently in all these cases been a most unexpected complication. Neither incision nor operative technic have been in any way unusual—though mastoid operations vary in extent and severity. The exposure and injury to the seventh nerve during mastoid surgery is usually known to the operator. As a matter of interest, its exit through the

*Read before the American Otological Society, Briarcliff Manor, June 19th, 1931.

stylomastoid foramen is in such proximity to the usual drainage exit of the parotid fistula that surgical repair of the latter is a matter of utmost caution. The sequence of parotid fistula, however, has apparently never been foreseen at the time of operation, nor caused by any known incident in its procedure. The mastoid wound seems to have closed normally, and following this a clear watery seepage occurred from a small and sometimes scarcely visible aperture at the lowest point of the mastoid scar. In some

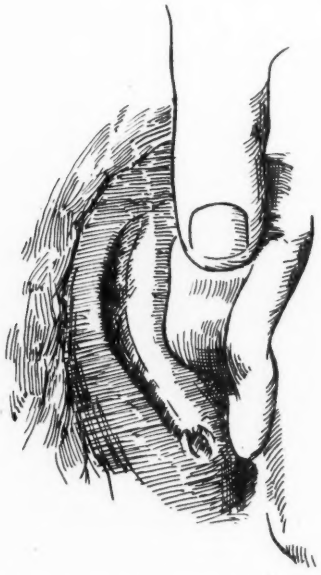

Fig. 1. Position of parotid fistula in the case reported.

of the cases, notably Combier's,[7] this did not appear until some weeks after complete closure. This may have occurred from original surgery, traumatism or by subsequent changes in the parotid gland itself or that portion near the mastoid tip.

Pain and tenderness have scarcely been associated, for its discovery has been more from annoyance at the presence of moisture coming down by the angle of the jaw and increased

by mastication and taking of tart foods which would excite salivary flow. The prodigious and uncheckable flow from an injured salivary gland is a matter of common experience, but these fistulæ, at least primarily, have evidenced but scanty secretion. In the case reported below, it was practically impossible to collect enough for laboratory test, either chemical or cytologic.

The anatomic picture and relationships of the parotid extending around beneath the lobule of the ear and resting upon the mastoid tip, in many cases closely adjacent to the insertion of the sterno-

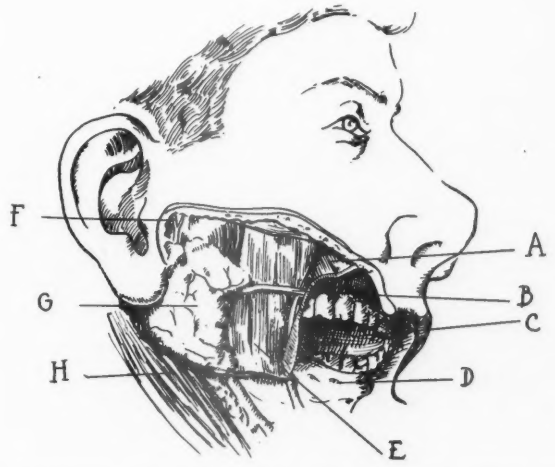

Fig. 2. Illustrating the parotid gland relation to the mastoid tip and muscle attachment.

cleido-mastoid fibers, further explain the likelihood of this fistula accident. Yet with this intimate relationship and the inviting path from the canal through the fissures of Santorini, parotid disturbance seldom occurs as a complication of external otologic surgery. Extensive mastoid surgery is a traumatic procedure, and an array of new adhesive bands and cicatricial rearrangement must occur. The delayed appearance of fistulous drainage could easily be due to subdermal retention pockets awaiting traumatism to link up with the parent gland or inflammatory tract ready to

break down from back pressure, on incidental block of main excretory duct. Such sclerotic changes in the terminal portions of lobular gland tissue would favor such fistulous breakdown.

The simple diagnosis of this discharge has initiated considerable laboratory effort. Schreiber[8] reviewed these in some detail, the psychic and acid food stimulation of secretion, polysaccharid splitting function, biuret test for mucin (found positive, though parotid not supposed to secrete mucin), administration of potas-

Fig. 3. Sodium iodide injection following the fistula tract to opening of Stenson's duct.

sium iodid for starch reaction, etc. In the case reported today, the lipiodol and sodium iodid injection for X-ray study of tract and appearance at Stenson's duct proved successful. Its procedure will be discussed by Dr. Kornblum of the University X-ray Department.

Methods for closure of fistula have been silver nitrate, chromic acid or actual cautery, Beck's paste, application of X-rays or

radium, injection of tincture of iodin, passing of ligature through tract for seton irritation, sometimes through duct and out of mouth, dissection of the tract and closure by plastic surgery. Sometimes several or combined methods have been necessary, and failure in efforts to control may readily result in increased flow and consequent annoyance to patient. It is obviously necessary to these procedures that the normal outlet through Stenson's duct be patulous and functioning. Cecil Wakely[9] advises opening down, section and avulsion of the proximal fibers of the auriculotemporal nerve, as first advocated by Leriche[10] in intractable

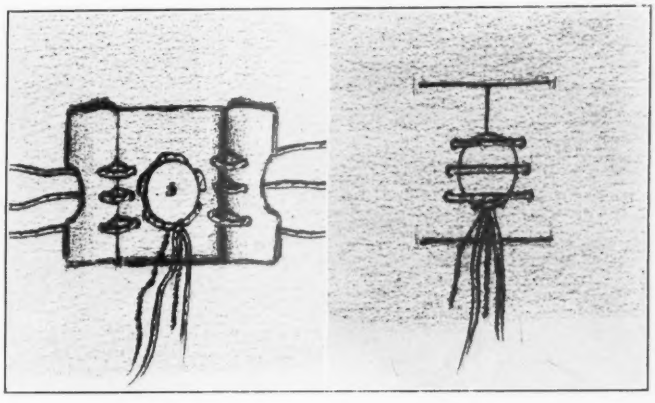

Fig. 4. Method for closing of fistula.

parotid fistulæ, causing not complete cessation of parotid secretion but a form of paralytic secretion. This may continue for some years, reduces secretion via Stenson's duct by one-half and stops fistulous discharge. His reported list included seven cases; several other authors have reported this method, and it should offer positive suggestion as a last resort.

Secretory stimulation to the parotid gland comes through the cerebrospinal and sympathetic nervous system, the former represented by the auriculotemporal from the second division of the fifth. The surgical section of this nerve does not far differ in effect from the deep X-ray treatment. Holman[11] states that treat-

Fig. 5. Position of auriculotemporal nerve posterior to the superficial temporal artery. After Wakeley in The Lancet, July, 1928.

ment by X-ray may dry up the secretion from the gland and stop the fistula but cites the danger of fibrosis and possible interference with the function of the facial nerve as contra-indication.

REPORT OF SPECIAL CASE.

As general medical conditions, associated pathology and laboratory detail present but little that is significant in relation to this local condition, the minutiæ of case history and unnecessary detail will be omitted.

The patient, Mr. F. R., a man, 73 years of age, was referred through another patient on August 19, 1930. He was an active man, an enthusiast on baseball, and came because of annoyance, not suffering, from a slight, rather oozing type of discharge through a minute fistula at the lowest edge of a former mastoid scar. This fistulous opening was on a level with the lowest portion of the ear lobe and about a centimeter behind it. He reported an

extensive mastoid operation by another physician a year and a half before. The drainage from the fistula had apparently followed rather imperceptibly the healing of the mastoid fissure. The details of ear and mastoid conditions prior to the first operation obtainable from the patient were meager—he simply presented with a perfectly good tympanic membrane, smooth enough to present a good light reflex, and creditable mastoid scar completely healed except at this point. His former surgeon was absent from the city at the time, but on return was very co-operative, described operative indications, extensive breaking down of cell elements throughout the entire mastoid, dura exposed through the tegmen antri.

In his words, "the convalescence was uneventful, and after the wound had entirely healed the salivary fistula became noticeable. This I unsuccessfully tried to close up by the use of eschoratics. I am unable to explain the occurrence, particularly as the parotid was not exposed when the tip of the mastoid was cleaned out." This treatment had probably in a measure been successful because when the patient came under present observation the discharge was not sufficient to collect for laboratory examination, and diagnosis was doubtful.

The preliminary studies of this patient were complicated by delay in obtaining these earlier details and confirmation of the previous salivary indications. The analysis of the case had to be made upon the immediate conditions presenting. The patient was very deaf on both sides, had a bad deflection of the septum, poor frontal and antral transillumination, secondary sinus and nasopharyngeal symptoms. The man was sent to the University X-ray Department for mastoid and sinus studies, and these were followed by lipiodol and sodium iodid injections, which will be discussed by Dr. Kornblum, associate in the department. The first and second X-ray reports were as follows:

August 23. "The roentgen examination of the right mastoid of F. R. shows that most of the cells have been removed. The lateral sinus is visible. I cannot see any break in the bony wall that connects with the nasopharynx. The anterior plate seems to be intact. There are considerable hypertrophic changes involving the fourth and fifth cervical vertebræ. Further examination of the sinuses and neck shows the following: Frontal, hazy on the left

side, probably clear on the right. Ethmoid, possible slight thickening of the cell walls on both sides. Maxillary, slight thickening of the mucous membrane lining, especially on the left side. The injection of the opaque solution into the sinus tract shows that the sinus communicates with Stenson's duct."

August 27: "We have made another roentgen examination of the sinuses of Mr. F. R. The injection of the sinus tract with lipiodol was not as successful as when sodium iodid was used. Only a few globules of oil are to be found in the sinus and Stenson's duct."

This would seem to have satisfactorily demonstrated the salivary tract and eliminated other fistulous complications.

Various office procedures were attempted in efforts for closure, silver nitrate, trichloracetic acid, chromic acid, galvano-cautery, and curettage. They only seemed to make matters worse. On September 2, 1930, at the Lankenau Hospital, with the assistance of Dr. Whiting, and under 1 per cent novocain anesthesia, the tract was exposed as far as seemed safe with proximity of the facial nerve, vigorously curetted, and closed by purse-string ligature and overlying plastic. The man seemed too feeble to take the risk of general anesthesia at this time.

Recovery was uneventful—but in the next few weeks slight dribbling appeared about five times and live cautery was immediately used. The patient went to Florida for the winter months, encountered the unfavorable weather of the past season and returned March 13th with trouble on the opposite side—an acute suppurative otitis media and tender, slightly edematous mastoid area. X-ray demonstrated definite involvement. His general health was not good. Medical examination reported an advanced sclerosis of the peripheral vascular system with hypotension, a double cardiac insufficiency and a low grade cystitis. This could not have been an entirely new situation and is quoted because of its possible significance as a background for the accident change in the other otitic field. Further laboratory studies demonstrated that the man was not a good mastoid operative risk. The parotid history in connection with the right ear further discouraged surgery on the left.

Therefore, under light administration of nitrous oxid and oxygen, a modified attic drainage operation, described briefly in the discussion following the middle ear symposium in the last session of the society, was done and mastoid symptoms were aborted, a dry ear obtained and no further loss in hearing noted.

The patient weathered this attack with no recurrence in the former fistulous opening of the right side, made a good recovery and registered his customary attendance at the baseball game.

Conclusions have already been given as a theme of this paper. It seems incredible that the list of reported cases should be so small, and it seems most likely that many of such in slight degree have occurred, and either spontaneously healed or readily responded to treatment.

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LVIII.

AURAL ACUITY AND BRAIN LESIONS. 1. AUDIOMETRIC STUDIES.*

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BOSTON.

In order of importance, the human ear is the foremost of all receivers of sound. This "organ of hearing," as it is sometimes called, has a marvellous range of sensitivity; it can appreciate vibratory frequencies varying from about 20 to 20,000 double vibrations per second, and can also distinguish changes in intensity over a very large range. The ear with its associated nerve endings has also remarkable powers of analysis, being able qualitatively to resolve a complex note into its several components, and to discriminate between a number of different sounds at the same time.

Ohm formulated a fundamental law in his statement that the ear recognizes as pure tones only those due to simple harmonic vibrations, and that it resolves any other complex vibration into its harmonic components, perceiving them as a summation of pure tones. This amounts to saying that the ear is capable of analyzing a complex tone into an harmonic series of simple tones. As everyone is aware, the ear is particularly sensitive to that characteristic of a sound which we call quality, which depends essentially on the number, intensity and distribution of the harmonic components into which a sound can be analyzed. Notes of the same pitch from a tuning fork, a violin, etc., to the ear, are entirely different in quality and are instantly recognized.

Some time ago a study was undertaken by us to determine standards or criteria of performance in various groups of persons who were regarded as having hearing powers above the average,

*Read at annual meeting of the American Otological Society, June 19, 1931.

†From the Evans Memorial and the Peter Bent Brigham Hospital.

either as the result of superior health or of a specialized training influencing the aural acuity. All members of the several groups were selected on the basis of absence of any demonstrable aural disease or impairment. The real goal was the standardization of a technic of observation which would permit the determination of significant relative acuity measurements and of quality interpretations under the usual conditions of clinical practice. In the collation of large amounts of data, care must be exercised both in recording and in interpreting. The average is but a mathematical quantity defining a linear relationship. The normal, on the other hand, is that individual whose departure from the average assumes only insignificant proportions.

Movement and sensation are the two extremes of the processes of human life by which the individual is brought into direct relation with the outer world. Movements are always objective in character and can be studied directly by external observation. Sensations are invariably subjective, and can be analyzed directly only by introspection; they are indirectly inferred from the expressional movements which are their external concomitants. Thus the messages from the different organs to the centers may definitely cross the threshold of consciousness and give rise to distinct sensations which differ in frequency and intensity. But very few of the impressions that reach us from the outer world, or from our own body, enter completely into consciousness, because the attention can be focused upon only a small part of the impressions received at any given time.

The apperception and interpretation of sound depends upon the aural mechanism in all its parts and upon the function of the central nervous system. With the intimate anatomic associations intrinsic in this system, the query naturally arises as to how far aural acuity would be affected by morbid changes with increased pressure levels inside the skull. The corollary is the possibility of interpreting such changes in terms of the actuating cause and thus aiding in the diagnosis and possibly localization of brain lesions.

Through the generous courtesy of Dr. Harvey Cushing, contact was established with the very large group of patients from his serv-

ice at the Brigham Hospital. It is the author's privilege to express his great indebtedness to Dr. Cushing for his unfailing kindness, his sustained interest and his liberal provision of every facility that could further the study. The study is now well into the second year of activity, and a sufficient number of cases have been studied to warrant at least preliminary presentation. It was Dr. Cushing's suggestion that each case admitted to his care be examined as far as was possible both before operation and again before leaving the hospital. This program has been carried out, and this thesis is thus based on such an examination of about 300 consecutive cases, unselected but grouped for study. The grouping has followed Dr. Cushing's practice, and the facts as presented are arranged into twelve groups based upon anatomic arrangement.

At the time of observation, each case was examined locally by an aural speculum, the audiogram determined by the No. 1-A audiometer (Bell Telephone Laboratories, Inc.), and the curve of bone conduction delineated by the Western Electric's D-80904 bone conduction receiver. The No. 1-A audiometer is essentially a vacuum tube oscillator, equipped with the necessary controls for regulating the frequency and output. The oscillator generates single frequency oscillating electric currents which produce tones in the specially designed receiver. By means of the controls tones of any one of the several pitches at octave or half octave intervals may be produced, and the intensity of these tones may be varied over a wide range.

With the audiometer in question, the pure tones of the oscillator are transmitted to the ear through a specially designed microphone. Pressures can be varied at will by the operator, and the threshold or pressure preception of the subject determined for each frequency. The technic followed in making these observations and measurements, with but minor exception, was devised by Bell Telephone Laboratories, Inc. The Weber test was carried out with the bone conduction apparatus, four points of contact being used, namely, the vertex, forehead, bridge of the nose and point of the chin. In addition, in many of the cases the Rinne test was performed, the quality acuity determined over a range

of the machine, a binaural examination carried out in suitable and suggestive cases and the history pertaining to the ears, cochlear and vestibular, compiled.

The composition of the several groups for the study of which opportunity has presented can be presented most compactly in tabular form.

TABLE I.

BASIC DATA.

Group Brain Lesion	Av. Age,	Male	Female	Patients	Retests	Total Grams
I. Frontal tumor.....	33	4	5	9	5	14
II. Pituitary tumor.....	39	30	27	57	14	71
III. Parietal tumor.....	43	7	2	9	6	15
IV. Temporal tumor....	47	10	1	11	1	12
V. Occipital tumor.....	41	6	4	10	5	15
VI. Acoustic tumor.....	43	14	21	35	20	55
VII. Cerebellar tumor, midline, lateral....	19	11	9	20	10	30
VIII. Arachnoiditis	30	2	8	10	7	17
IX. Multiple sclerosis....	36	3	1	4	1	5
X. Cerebral tumor.....	37	35	47	82	19	101
XI. Intercranial tumor...	35	16	14	30	8	38
XII. Miscellaneous	37	6	8	14	1	15
Total or average.....	37	144	147	291	97	388

This table includes only the cases that have been examined personally and of course does not represent Dr. Cushing's total values as to types of disease or to the pathologic diagnosis of the tumor masses. The miscellaneous group was composed of several spinal tumors, cases of tic, tuberculosis of the skull, labyrinthitis, epilepsy and Paget's disease. The children largely came into the group of intracerebellar tumors. The majority of the examined cases were in their middle range of life.

As to the data that can be derived from the histories regarding the acoustic nerve can be arranged for graphic illustration in Table II:

TABLE II.

OBSERVATIONS AND REPORT.

Group	Deafness	Tinnitus	Ostoscopic Exam.	Dizziness	Vertigo	Caloric Tests
I.	0	0	negative	3	0	0
II.	4	0	"	3	0	0
III.	2	0	"	1	0	0
IV.	0	0	"	0	0	0
V.	0	0	"	3	0	0
VI.	35	35	"	30	25	35
VII.	0	0	"	2	1	0
VIII.	0	0	"	0	0	0
IX.	0	0	"	0	0	0
X.	3	1	"	1	0	0
XI.	1	1	"	0	0	0
XII.	2	1	"	1	2	0
Totals....	47	38	negative	44	28	35

With the exception of Group VI (acoustic neuromas), the infrequency of report in the patient's history of eighth nerve involvement, the surprise of the patient at the finding of upper tone area deafness of more than 10 per cent practically without exception and the changes in the bone conduction curve constitute a group of extremely striking phenomena. Where the air and bone conduction curves approach the normal, the negative findings in the brain and the paucity of other definite neurologic signs, it is striking. The gait changes or other primary cerebellar symptoms have not been listed in the table. The few cases of deafness noted in the table outside of Group VI were incidental and did not have a bearing upon their admission. The two cases of retracted drums were in other groups than the acoustic tumor. Omitting the acoustic neuroma tumors, less than .03 per cent of the entire group show any degree of deafness or tinnitus. There were no positive caloric tests in any of the groups except the acoustic tumor group. Dizziness and vertigo seem to be more associated with headache and pain in the head.

Following is a brief description of each of the twelve groups with protocols of illustrative cases:

Group I represents nine cases and fourteen audiograms; the first audiogram a composite curve for air and bone conduction.

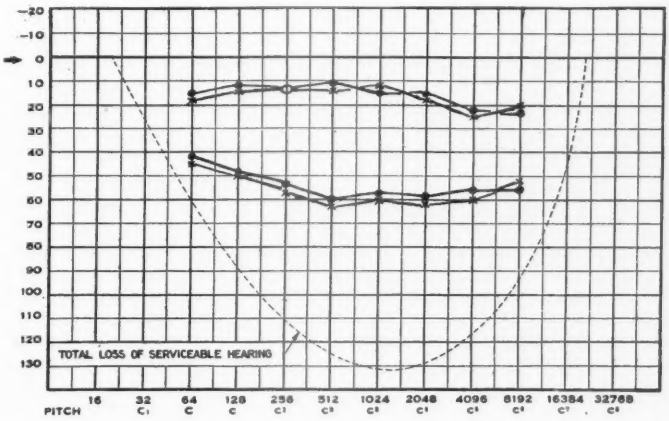


Fig. 1, No. 1, Group I. Frontal tumor; composite curve for air and bone conduction.

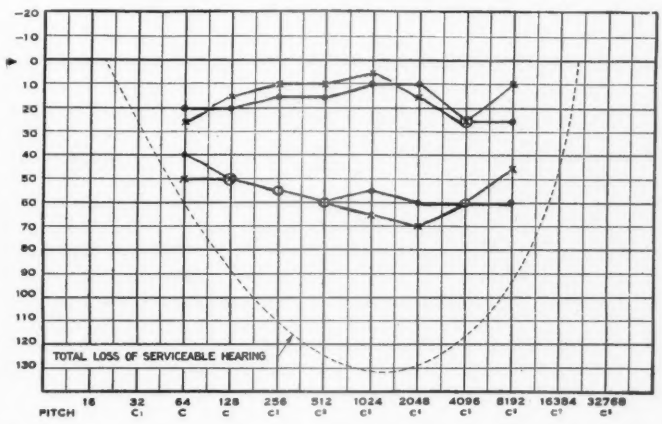


Fig. 2, No. 1a, Group I. Case 1-7-291. Frontal tumor, gram before operation.

The air conduction has a tendency to fall off at 2048 dv., while the apparent tendency with the bone conduction is to be somewhat lengthened at the lower levels and to distinctly lengthen at 512 dv., both ears apparently paralleling each other. The Weber test was generally not referred. The nine cases showed otoscopically normal drum membranes without ear involving history. An illustrative audiogram of a verified tumor of the frontal lobe is shown, showing the tendency of the curves with the lengthening of the bone conduction throughout the eight octaves.

Case 1-7-291. The chief complaints were headaches for two months and failing vision and diplopia for one month. Under a summary of positive findings were noted: increasingly severe suboccipital headaches in the morning during the last two months, with slight blurring of the vision during the last month. There had been diplopia for three weeks and questionable visual hallucinations for two months. The definite findings were a slight bilateral suboccipital tenderness and apparently complete bilateral anosmia. The eyes showed bilateral acute choking of 5-D. without secondary atrophy, the eyes were myopic. A slight reduction of visual acuity was present without any field defects, however, there was a slight bilateral abducens weakness, more marked on the left side. Present also was a slight lower facial weakness while the abdominal reflexes were absent on the left side. An obvious mental impairment was noted as well as a lack of mental concentration.

Group II. This group consisted of pituitary tumors, the series being composed of chromophobe or chromophile adenomas, with or without acromegaly or giantism. The composite gram is of fifty-seven cases and seventy-one audiograms.

The air conduction shows a tendency to fall off at about 2048 dv., while the bone conduction begins to distinctly lengthen at about 256 dv., and also shows a tendency to return upward at 8192 dv.

Three rather typical audiograms and short case protocols are shown in this group.

Only one case showed a slightly retracted drum membrane, which was apparently of no moment. The entire group showed no tuning-fork changes that were at all suggestive of disease. There seems to be no apparent change in the hearing curve following operation.

Case 2-17-111 had as chief complaints headaches and loss of vision in one eye. There had been a history of failing vision over a period of twelve months which was noticed particularly in the temporal field and

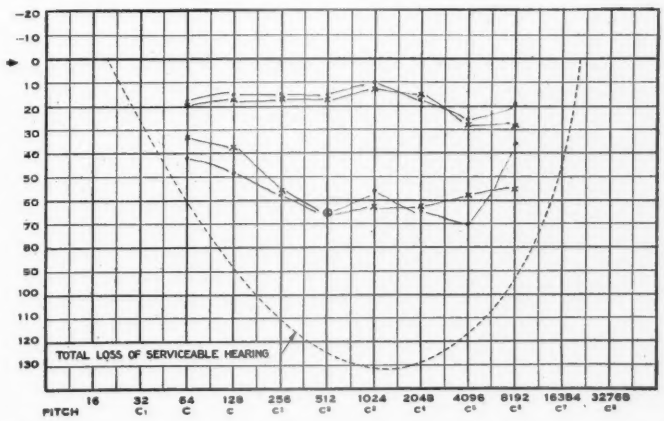


Fig. 3, No. 2, Group II. Pituitary tumor; composite curve for air and bone conduction.

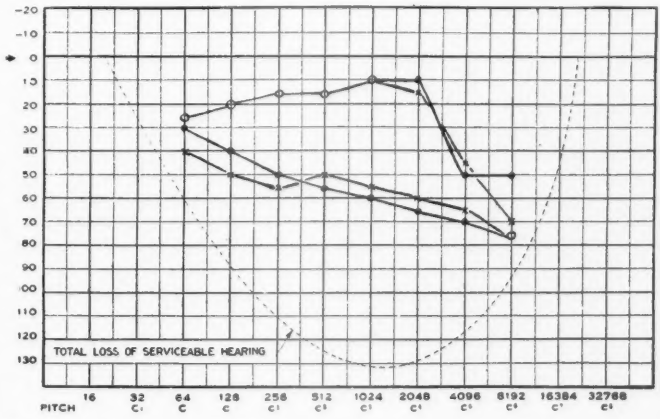


Fig. 4, No. 2a, Group II. Pituitary tumor. Case 2-17-111; chromophobe adenoma.

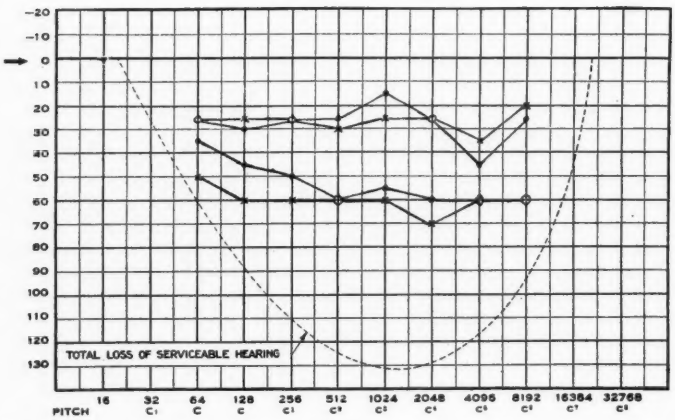


Fig. 5, No. 2b, Group II. Pituitary tumor. Case 2-70-307; chromophile adenoma.

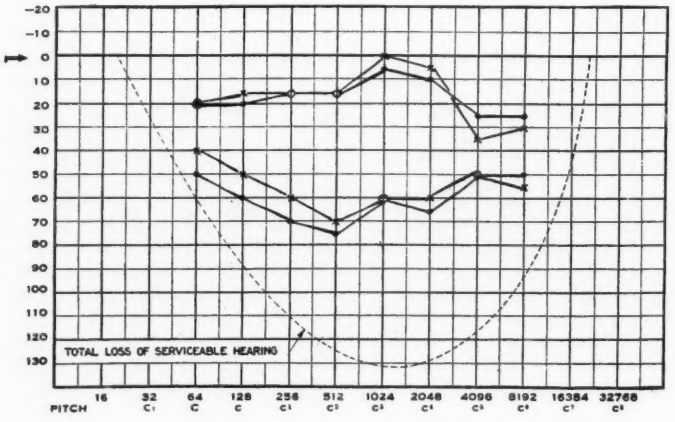


Fig. 6, No. 2c, Group II. Pituitary tumor. Case 2-52-82; suprasellar cyst.

rapidly progressing amounted at admission to complete blindness in one eye and could just discern light from dark in the nasal field in the other eye. Also were noted mild bitemporal headaches, with slight alleviation of the intensity since that time. Some recent loss of weight being noted associated with a good appetite. Objectively the case showed bitemporal hemianopsia, a bilateral primary optic atrophy which was most marked in the left eye. X-ray showed enlargement of the sella turcica and the basal metabolic rate was —8%.

Case 2-70-367 complained of headache and had enlargement of the features. The headaches were of five years' duration while the acromegalic changes were noticed first about one year ago. Menstruation ceased at about the same time. There were typical acromegalic changes of the face, hands and feet, the basal metabolic rate being plus 5%. The perimetric fields showed contraction and the sella turcica was expanded.

Case 2-52-82 complained of a gradual diminution of vision over a period of three years, beginning first in the temporal field of the left eye and progressing to almost complete blindness in that eye with a temporal hemianopsia of the other. There had been a history of vertical headaches of many years duration, lessening, however, during the last two years. During the last year there had been loss of libido. Objectively he showed a bilateral primary optic atrophy and diminished pupillary reactions to light in the left eye.

GROUP III. PARIETAL TUMORS.

The composite gram of the nine patients and fifteen audiograms seem to show a tendency for the air conduction to be generally lower throughout and a slight rise at 1024 dv., then a distinct falling off from that point to the end of the scale. The bone conduction curve is apparently lengthened throughout, while the contour in general in the upper half of the scale shows a tendency to return upward.

The audiogram of the illustrative case shows very markedly drops of both air and bone conduction at the upper frequencies. The bone conduction, I might add, shows no tendency to conform to the normal bone conduction line. A fairly typical protocol was the following Case III-10-88, exhibiting unilateral sensory and motor Jacksonian attacks for a number of years with more recent headaches. The Jacksonian attacks consisted of twitching of the arm and neck, perhaps followed by numbness of that arm and side of the face, but there would be no loss of consciousness. Recently the numbness has become permanent. There had been slight weakness and dragging of the foot for several months, while there had been very severe right-sided headache during

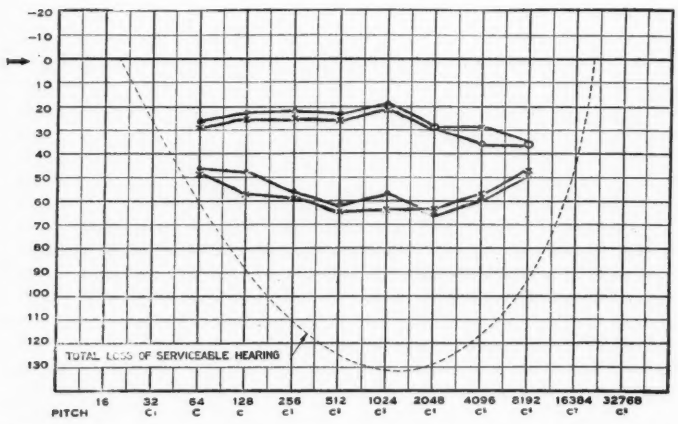


Fig. 7, No. 3, Group III. Parietal tumor; composite curve for air and bone conduction.

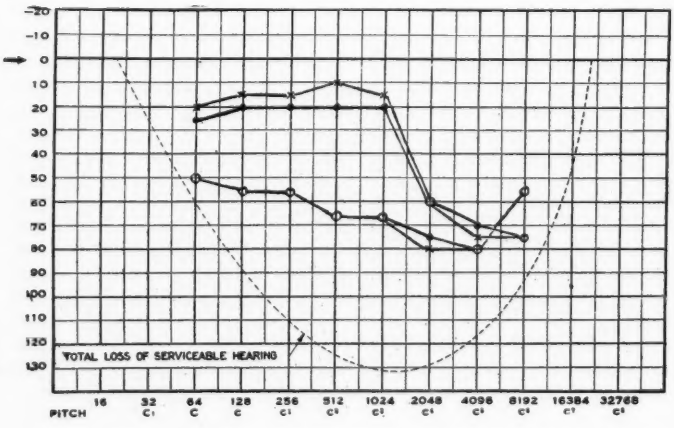


Fig. 8. No. 3a, Group III. Parietal tumor. Case 3-10-88.

several periods of time. Objectively the patient showed slight suboccipital tenderness with acute choking of 5 D. in one eye, but no measurable choking of the other eye. A slight hyperesthesia to thermal sensation over one side of the body was noted and a slight hyperesthesia to pinprick over one arm. Also a slight weakness of the dorsi-flexors of one foot and dragging of the foot while walking. Present also was an increase in the deep reflexes of one leg with an ankle clonus and a positive Babinski. Astereognosis of one hand and a very marked loss of muscle sense was present in the hand and in the foot. The X-ray showed a localized thinning of the parietal bone, and although the patient is left handed there was no aphasia noted as being present.

Group IV is composed of eleven cases and twelve audiograms.

The composite group curve shows a tendency for the air conduction to rise slightly at 1024 dv., then to drop off at once with a suggestion of again recovering at about 8192 dv. The bone conduction curve shows a regular lengthening at the lowest frequencies, then paralleling from 256 dv. to 1024 dv., next lengthening still more markedly through 4096 dv., and with a tendency to rise slightly at 8192 dv. The general contour above 2048 dv. was toward normal but at least twenty sensation units below that level.

In this group it might be noted that the tumor may be on either side, as far as the ear history is concerned. The eleven cases gave no suggestive ear findings and the case protocols showed characteristically the following points:

Case 4-1-23. The chief complaint noted a gradual increasing nervousness with an inability to concentrate easily, and a jerking of one of the members of the body for some period of time was noted. The headaches were frontal, over the vertex and into the occipital region, unassociated with nausea. There had been a history of left hemihyperesthesia in the upper arm which was greatly relieved by a partial parietal decompression in 1929. Objectively the scar showed a right parietal bone flap in the hinge of which there was a large and excessively tense and bulging decompression evidently containing herniated brain. Bilateral choking of the discs of 3-D., and a left homonymous hemianopsia were noted to a rough test. Present also was a marked left lower facial weakness of central type, a left hemiparesis, hemihyperesthesia and astereognosis most marked in the upper extremity.

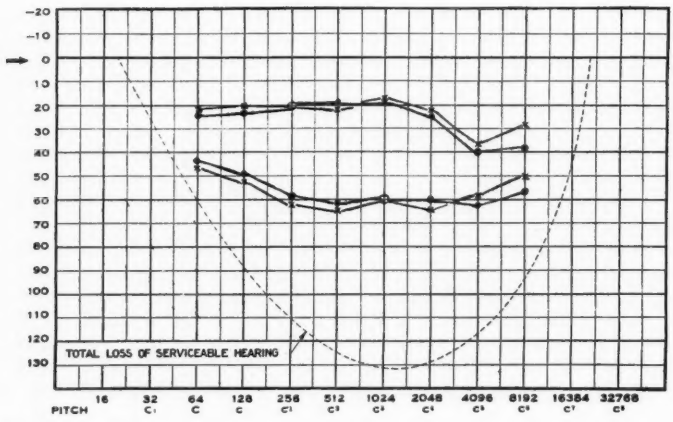


Fig. 9, No. 4, Group IV. Temporal tumor; composite curve for air and bone conduction.

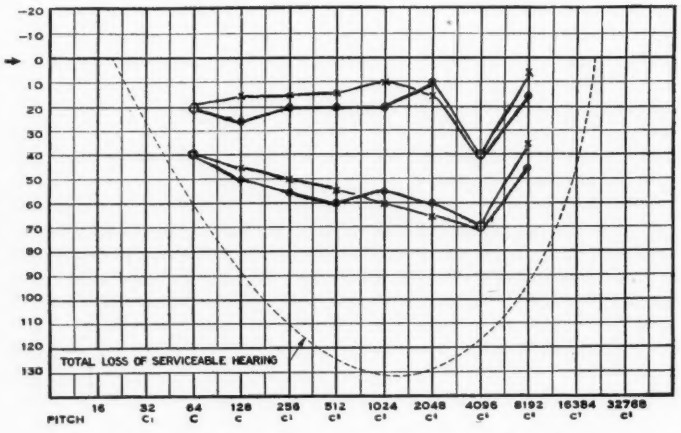


Fig. 10, No. 4a, Group IV. Temporal tumor. Case 4-1-23.

Group V. The occipital tumor group was composed of ten patients and fifteen audiograms.

The composite curve shows again a slight rise at 1024 dv., then a direct drop throughout the rest of the scale in the air conduction, while the distinctive point, perhaps, regarding the bone conduction curve is the straightness between 512 dv. and 1024 dv., together with the general tendency upward through the rest of the scale. Throughout the lengthening of bone conduction is to be noted, both ears showing equal and uniform deviations.

A typical audiogram of this group with the case protocol is as follows:

Case 5-1-199. The chief complaints were of very severe attacks of headache, accompanied by vomiting. The objective findings were a well healed scar of a right, very markedly bulging temporo-occipital bone flap from which the bone had been sacrificed, the decompression being tense. A slight choking in both eyes was seen without measurable elevation, also a slight lowering of visual acuity with a left-sided homonymous hemianopsia. Objectively were noted slight diminution of thermal sensation over the left side of the body, and astereognosis in the left hand, with a marked diminution in deep muscle sense of the left hand and the left foot. The left abdominal reflexes were absent, while a slight bilateral and equal ataxia on the finger to the nose test was present. This patient was naturally left handed, but showed no sign of aphasia.

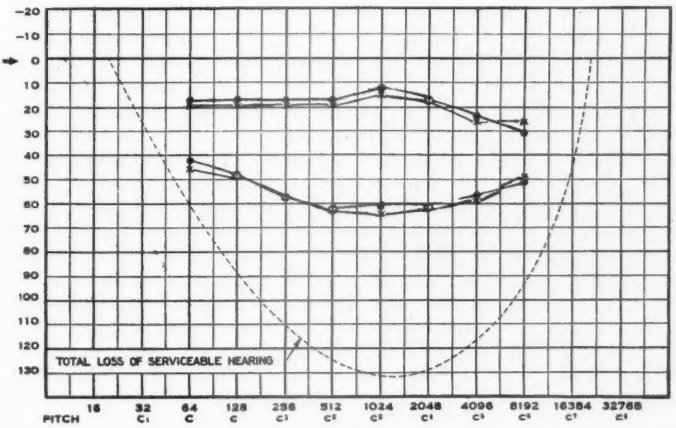


Fig. 11, No. 5, Group V. Occipital tumor; composite curve for air and bone conduction.

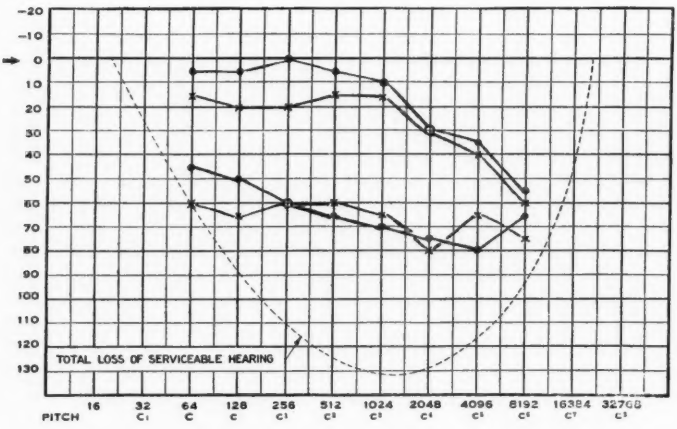


Fig. 12, No. 5a, Group V. Occipital tumor. Case 5-1-199.

Group VI, extracerebellar tumors, acoustic neuromas and pontine angle tumors), show the following composite curve:

The air conduction, as one would expect, is greatly depressed throughout the scale. More so on the side of the tumor, except the pontine angle tumors, and they show a very nearly normal curve. The bone conduction shows lengthening also, as well as a toboggan-like tendency, except possibly at the very high limit of frequency. The air and bone conduction curves tend to run together, paralleling, in fact, more than in any of the other of the groups. These thirty-five cases all showed positive caloric reactions, complained of deafness, tinnitus, dizziness and vertigo. The Weber test was to the deafened side, the four points bringing out several interesting observations. The quality curve showed that the deafness was not of the middle ear type, and the binaural hearing also showed extremely interesting and valuable points. All through this group the left ear seemed to be somewhat more keen than the right ear, both in air conduction and in bone conduction.

A typical case protocol would show in all probability the following points: The entrance complaint would probably be dif-

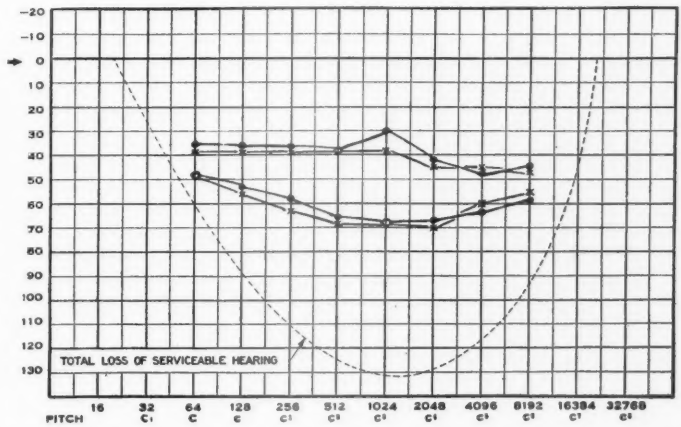


Fig. 13, No. 6, Group VI. Acoustic tumor; composite curve for air and bone conduction.

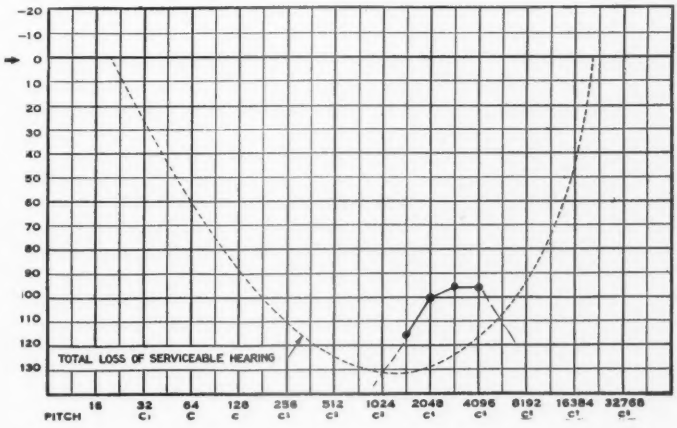


Fig. 14, No. 6a, Group VI. Acoustic neuroma. Case 6-45-4, showing a small island of remaining hearing in the left ear.

ficulty in hearing, together with tinnitus and more or less blindness. The history would be of long standing and the positive findings would embrace choking of the discs, usually bilateral, nystagmus together with vertigo and gait changes. The caloric tests consist of irrigating the ear canals with water at 40° C. for a two-minute period when pathologic, shorter if not diseased. The time of active nystagmus appearing, if any, and the manner of p. p. are the points noted. There is generally facial weakness with a loss of the corneal reflex and some cerebellar changes of the control of the arms and the legs. Generally this group of tumors do not seem to make the individual as sick or helpless as in several of the other types of tumors.

The illustrative audiogram is that of the most deafened individual that I have ever had the opportunity of examining, outside of those with congenital loss. I found that this case had absolutely no bone conduction that I could detect, no binaural ability or qualitative hearing whatsoever. It was not due to lack of mental ability.

Group VII.—The intracerebellar tumor group shows twenty patients and thirty audiograms, most of them being children, with the tumor mass being either midline or lateral in placement. The composite curve shows a tendency to upward bowing of the air conduction, the peak being at 1024 dv., while the lower and upper extremes are about on the same plane. The bone conduction curve bows likewise, but downward, the peak at 512 dv., with a slight lengthening of the bone conduction throughout. At the upper level there is a tendency to return to the normal but still remaining distinctly lengthened in time.

A rather typical audiogram of the series. The case protocol is as follows:

Case 7-20-51. The complaints were found to be ataxia, headache with and without vomiting, some stiffness of the neck and tenderness, an occasional diplopia and diminution of vision of about eight months duration. The objective findings were a cracked-pot sound to percussion over the skull, associated with a soreness and stiffness of the neck. A bilateral choking of the discs, 5-D. was noted superimposed on secondary atrophy that was most marked on the right side, the right eye being blind to a rough test. Nystagmus of the right eye to the left and upward (coarse)

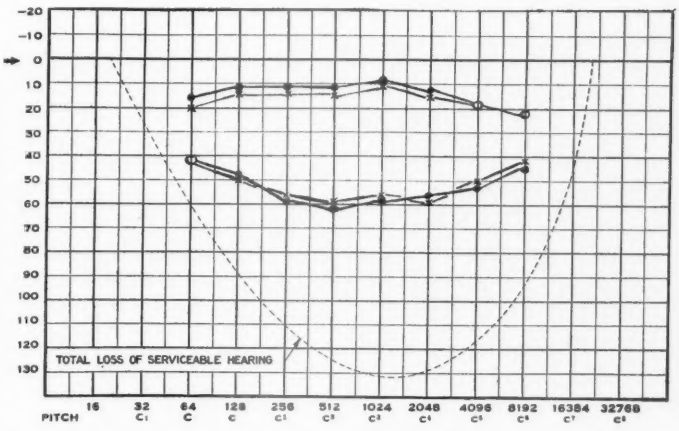


Fig. 15, No. 7, Group VII. Intracerebellar tumor; composite curve for air and bone conduction.

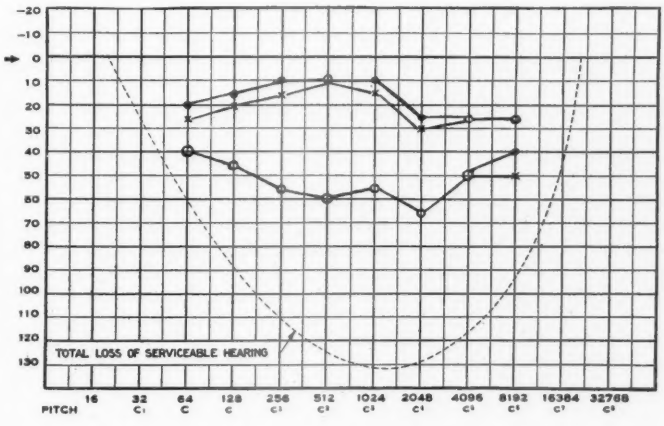


Fig. 16, No. 7a, Group VII. Intracerebellar tumor. Case 7-20-51.

with external and upward deviation of the right eye. Present also were a positive Romberg and a tendency to fall to the right when walking, cross ataxia of the arms and legs, as well as diminished deep reflexes throughout the body and especially so on the right side of the body. The helplessness of these cases is very marked.

Group VIII was composed of ten cases and seventeen audiograms of arachnoiditis, which show the following composite group curve for both air and bone conduction:

The tendency in the air conduction seems to be a peaked upward bowing at 1024 dv., the upper level of the bow end being somewhat depressed. The bone conduction curve shows a tendency to a flatness from 256 dv. to 4096 dv., with an upward tendency toward normal at 8192 dv., although throughout the time is lengthened.

As an illustrative audiogram, I insert the following extreme case:

Case 8-6-29. The point of interest is that the pressure is sufficient to effect the hearing in the upper level in both ears equally. This patient did not complain of his ears in any way and was very surprised that the curve was not normal or rather not of the average. He felt quite sure that something must be wrong with the battery. His chief complaint was severe headache, frontal and occipital. This type of a case may have nausea and vomiting accompanying the headaches. They may also have considerable difficulty in walking; choking of the discs is often found.

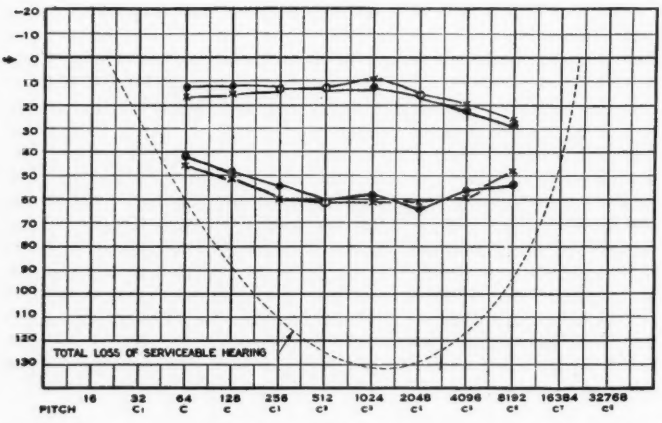


Fig. 17, No. 8, Group VIII. Arachnoiditis; composite curve for air and bone conduction.

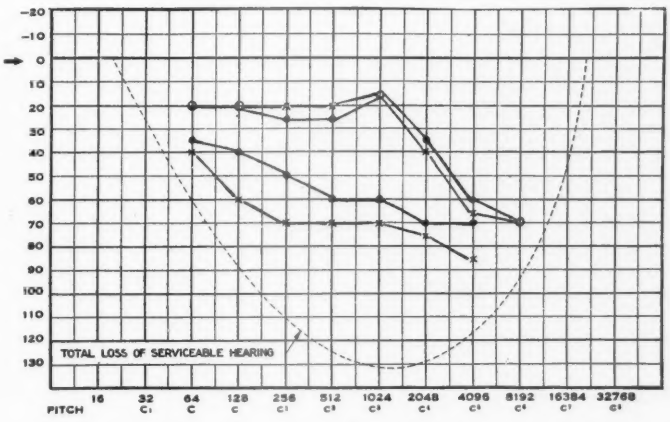


Fig. 18, No. 8a, Group VIII. Arachnoiditis. Case 8-6-29.

Group IX, multiple sclerosis, includes four cases and five audiograms for this group:

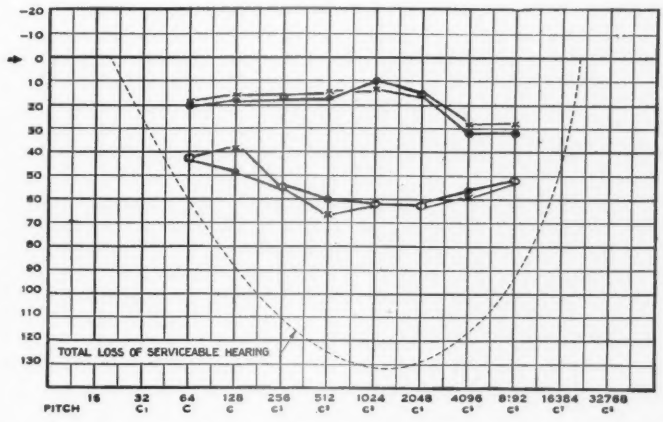


Fig. 19, No. 9, Group IX. Multiple sclerosis; composite curve for air and bone conduction.

The air curve seems to have a tendency in all the cases to have a sharp drop at 2048 dv., while the bone conduction does not seem to be so pronounced at that point. Throughout, the bone conduction time is lengthened, but very slight indeed at the lower frequencies.

An audiogram of the case is given in Graph 18.

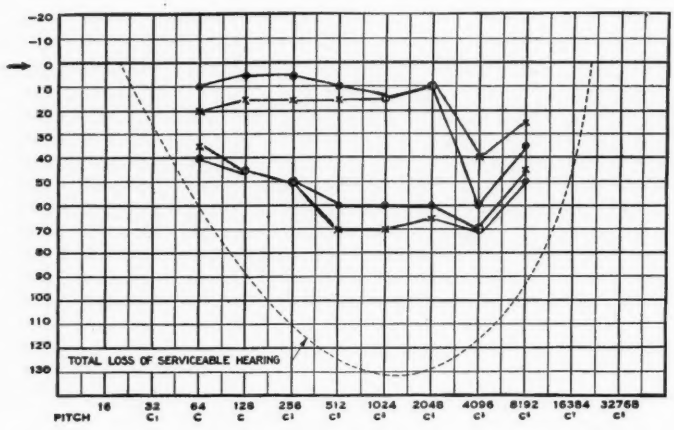


Fig. 20, No. 9a, Group IX. Multiple sclerosis. Case 9-2-27.

Case 9-2-27. The patient complained of peculiar migratory paralysis of the extremities and ataxia with transient attacks of dysarthria and dysphagia together with dull vertex headaches unassociated with vomiting. Also present were incontinence of the urine and fæces with a history of an occasional diplopia. Objectively the case showed an interesting flat, monotonous speech with pallor of both discs. Noted also were a coarse, rotary nystagmus in all directions with a slight right abducens paralysis. There had been a slight deviation of the palate to the right. While paralysis of both lower extremities and the left upper extremity associated with hyperesthesia, astereognosis, pathological reflexes, increased deep reflexes and absent abdominal reflexes were noted.

Group X, cerebral tumor, includes eighty-two patients and one hundred one audiograms.

The composite curve, seems to show a general tendency to depression in the air conduction, especially above the speech area. The bone conduction is practically normal to 512 dv., then shows a tendency to lengthen, but in general it would seem to follow the normal contour line.

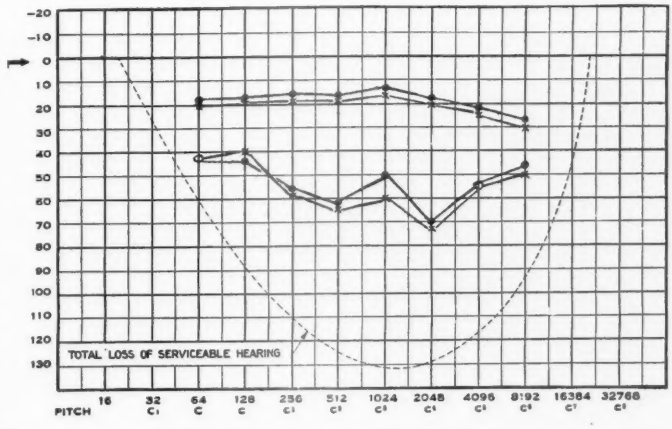


Fig. 21, No. 10, Group X. Cerebral tumor; composite curve for air and bone conduction.

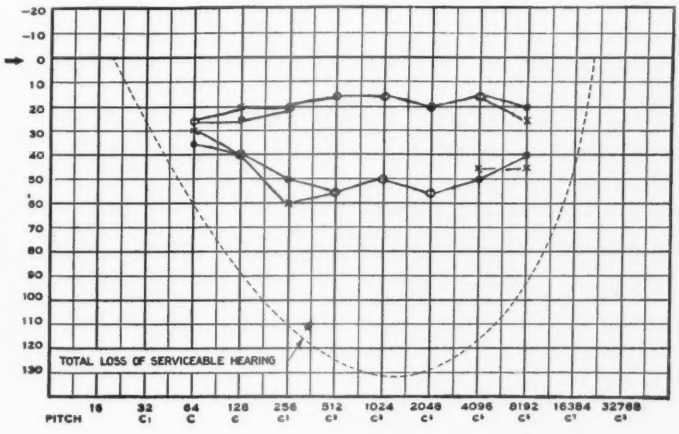


Fig. 22, No. 10a, Group X. Cerebral tumor. Case 10-54-161; "suspect" tumor; final diagnosis, manic depressive insanity.

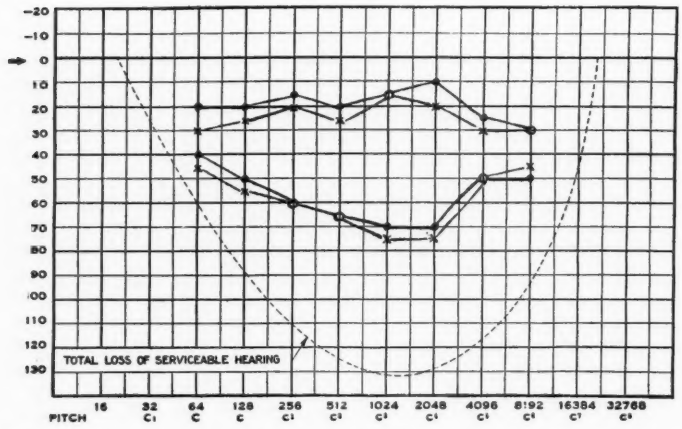


Fig. 23, No. 10b, Group X. Cerebral tumor. Case 10-53-236; "suspect" tumor; final diagnosis, manic depressive insanity.

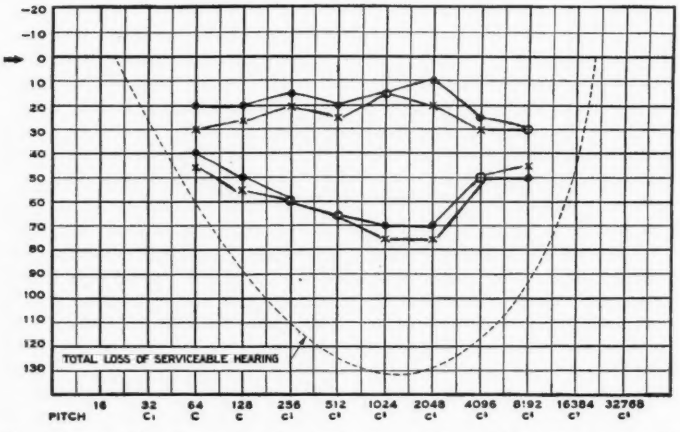


Fig. 24, No. 10c, Group X. Cerebral tumor. Case 10-52-237; "suspect" tumor; final diagnosis, manic depressive insanity.

I desire to bring to your attention, under this group heading, several interesting audiograms. The first three "suspect tumor" cases proved at discharge to be manic depressive insanity. The three had not recognized their deafness, and you will note the

same tendency in their grams in air conduction and bone conduction. The fourth case is entered as a case protocol as follows:

Case 10-58-47 included a history of epistaxsis for one year, cramps in both calves as well as weakness in both legs for three months, tinnitus for three months, and failing vision with a history of dizziness and staggering of gait for several weeks duration. Objectively the patient showed a right facial palsy of central type and blood changes of a splenomyelogenous leukaemia. The ears showed slightly thickened, pale drums and more marked on the right side. It illustrates by audiogram what Alt first described in 1896.

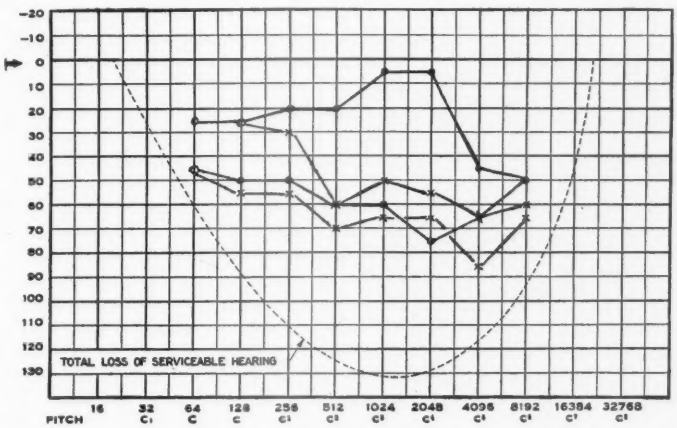


Fig. 25, No. 10d, Group X. Cerebral tumor. Case 10-58-47; "suspect" tumor; final diagnosis, splenomyelogenous leukemia.

Group XI, intracranial tumor, includes thirty patients and thirty-eight audiograms.

The composite curve of Group XI seems to show a general tendency for a slight rise at 1024 dv., then a distinct drop to the end of the scale in the air conduction. The bone conduction curve, on the other hand, seems to show a tendency toward normalcy up to 512 dv., then a regular lengthening of time to the end of the instrument.

The following two audiograms, taken about a year apart, at two different admissions, illustrate a patient who entered the hospital complaining of failing vision, headaches, vomiting and lassitude. He refused treatment and on re-entry, a year following,

was then totally blind. The patient also had at this time a positive blood Wassermann. The first audiogram is quite normal, the second shows a "dipper gap" at 4096 dv.

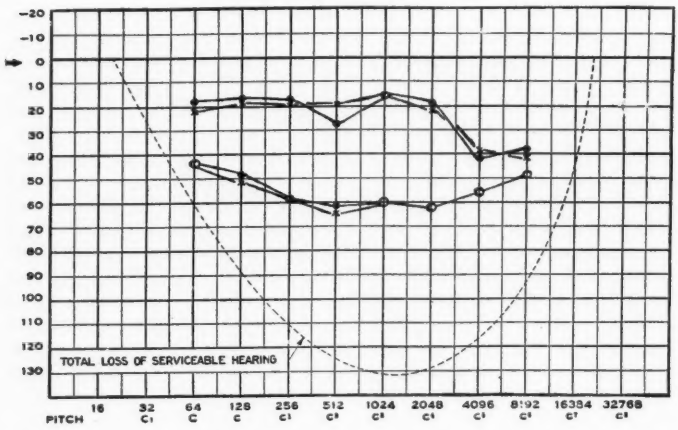


Fig. 26, No. 11, Group XI. Intracranial tumor; composite curve for air and bone conduction.

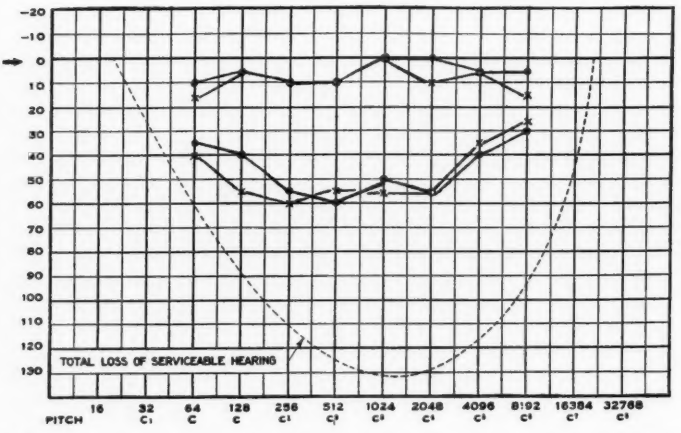


Fig. 27, No. 11a, Group XI. Intracranial tumor. Case 11-57-56, showing normal acuity curve.

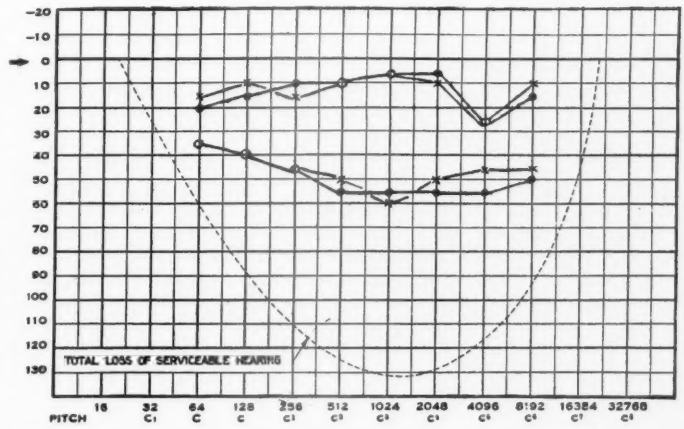


Fig. 28, No. 11b, Group XI. Intracranial tumor. Case 11-38-326, showing a dipper gap at 4096dv. Same individual as Fig. 27, audiograms taken a year apart. Wassermann positive.

Group XII, miscellaneous group of cases, representing fourteen patients and fifteen audiograms.

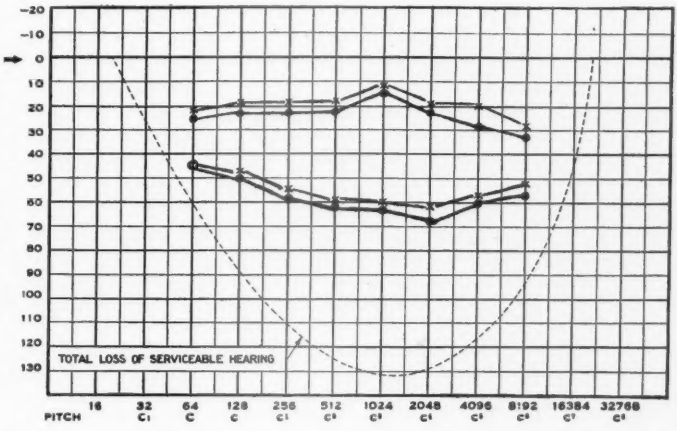


Fig. 29, No. 12, Group XII. Miscellaneous group; composite curve for air and bone conduction.

The composite curve seems to illustrate both in the air and the bone conduction curves that the hearing acuity is directly affected by the lowered metabolic level of the individual.

A gram of interest in this group is the following:

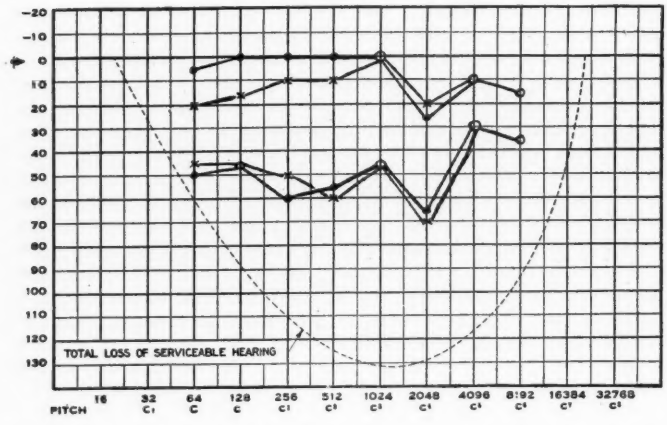


Fig. 30. No. 12a, Group XII. Miscellaneous group. Case 12-404; concussive deafness.

Case XII-404. A man who was unconscious of any deafness and who had been badly gassed and shocked by artillery fire. It is cited to confirm Fowler's opinion.

DISCUSSION.

The purpose of presenting this material at this time is to point out that audiometric study offers another excellent aid toward the establishment of a correct final diagnosis. We know that as the tones rise in the scale sensibility increases rapidly, and reaches its maximum in the frequencies between 1000 and 5000 dv., above which it again declines. Generally, we recognize that high tones are louder and more penetrating than low tones, and a very weak tone is only fully appreciable to the sound ear after a lapse of one to two seconds. As a class, tumors of the frontal area seem to show the least effect upon the ear. This may be explained by

the fact that the interpretative ability of the individual has been so depressed that the sound does not register in their consciousness.

CONCLUSIONS.

1. The results obtained by audiometric study of 291 cases of brain lesion are presented. These are divided into twelve groups. Nearly one-half the series have been studied both before and after operation.

2. The most striking single feature is the lack of report of deafness in the history and the demonstration by objective means of its presence. The allocation of the failure to the upper tones, superior to the speech area, is the patent explanation of this seeming contradiction.

3. The indications in the several groups suggest that careful audiometric study may prove to be of aid in the diagnosis and localization of brain lesions.

I desire to take this opportunity of again expressing my most sincere appreciation toward both Dr. Cushing and Dr. Rowe for their extreme co-operation.

416 MARLBORO ST.

LIX.

THE GRADUAL CHANGE IN THE OTOLOGIST'S CONCEPTION OF THE CONDUCTION AND PERCEPTION OF SOUND IMPULSES.

FRANCIS P. EMERSON, M. D.,

BOSTON.

The physical theory of the propagation of sound tones formulated by Helmholtz was accepted by otologists and also by all scientists up to the twentieth century.

During this time physics, biology, physiology and neurology were called upon to explain some of its problems. Whatever may be said about the molecular theory of Helmholtz or the biologic theory of Ewald in regard to sound conduction, the otologist has signally failed in utilizing these theories to correct the impairment of sound perception.

Has not this failure been due largely to the fact that we have been trying to explain nerve dysfunction in terms of physical obstruction? Sound tones may fail completely or partially to reach the perception center, but obstruction never causes nerve derangement unless pressure, irritation or long continued excessive sound tones coexist as additional factors. Neither does the relief of obstruction in any way affect the etiology of an intermittently progressive disease as that which characterizes the gradual loss in tone perception which otologists have to treat.

The failure to recognize an active and intermittent cause that is operative over a long term of years makes it difficult on physical or biologic grounds to explain when a negative Rinne commenced or what symptoms were present to indicate that nerve impairment was taking place as well as obstruction in the middle ear. Neither have we any explanation of the loss in tone perception in those cases of tubotympanic catarrh that completely clear up, at least on physical grounds, and yet this is the beginning of most of the cases of slowly progressive deafness. Furthermore, tinnitus ushers in and is intermittently present in all the catarrhal

middle ear cases when the tissue reaction is not sufficiently acute to cause any labyrinthine irritation. Clearly, some cause must be found outside the middle ear that may or may not be related to physical changes in the conduction apparatus, for, as has been shown, they are not always present.

In the last few years we have, as otologists, had to modify our views as to what constitutes obstruction in the conduction apparatus, which has left a large number of cases, where tone perception was being gradually lost, to be accounted for on other than physical grounds, as there was no evidence of tissue reaction in the middle ear. These cases will be discussed later on in this article.

Let us return for a moment to a consideration of the conduction apparatus. One of the early errors that was misleading in our understanding of the pathology of the middle ear was due to the misconception of the nature of a catarrhal process in that it was generally thought to be nonbacterial in origin. It would seem to be obvious that any excess secretion from a mucous membrane must be due to an irritant from either a mechanical, chemical or bacterial source. A mechanical cause could be easily eliminated, any chemical change that was constantly present in the secretions was never demonstrated, hence the reference must be that it was due to bacteria.

The old nonbacterial theory prevailed until Zuckerkandl, Hirsch and Uffenorde at about the same time proved that it was a low grade infection in which there was a serous infiltration of the mucosa which became swollen to ten or fifteen times its normal thickness. This intense congestion was followed by a serous effusion or exudate. Under these circumstances an intensely congested eustachian tube would be followed by a serous effusion into the middle ear, without the necessity of any negative pressure being present. A bacterial origin would then place a catarrhal process in the same category as that of any other infection that was followed by tissue changes in the middle ear.

We next come to the eustachian tube. This has been inflated and bougied since the beginning of otology, on the supposition that it was obstructed, although no chronic case in which tone

perception was being gradually lost was ever cured by such treatment or even improved.

At the Harvard Medical School 900 careful dissections were made by Faunce for teaching purposes, and only one case of obstruction was found, and that was near the mouth of the tube. When we consider that most of the cases in which there is a gradual loss in tone perception occur in connection with a catarrhal process, and that this process is such a low grade infection that it is seldom, if ever, followed by erosion or the formation of cicatricial tissue, we would not expect to find any obstruction. If any obstruction were found it would be in connection with a suppurative tubercular or syphilitic process. Such cases have seldom been reported.

In the passage of a bougie the catheter makes such an angle with the long axis of the eustachian tube that its point would be apt to impinge on its posterior wall. In such a case, by changing the curve of the catheter, or by using a larger bougie, most of these obstructions disappear. A classification of middle ear disease which is gradually being dropped, was known as the catarrhal adhesive type. In these cases the membrana tympanum was clear and there was no pathology to be seen in the tympanic cavity.

Reasoning on the theory of physical obstruction, it was supposed that the loss in tone perception must be due to an increase in the connective tissue stroma without involving the lining membrane. Here the basic principles of pathology should have shown us that no increase in the connective tissue could take place without an increase in the circulation and a round cell infiltration which would involve the lining membrane. No doubt this classification would have been discarded earlier, but the physicists had no other explanation to account for these very common cases. Indeed, some otologists went so far as to classify them all as cases of beginning otosclerosis, although there was nothing to justify such a diagnosis. Their etiology is clearly shown in a later consideration of these cases.

It is a common clinical experience that sound perception is better following a long continued purulent than a catarrhal process, and yet, on examination, we may find a rigid, thickened and

calcified drum membrane with a large perforation and the tympanum full of adhesions, or we may have a condition similar to a case that the writer has followed for ten years, in which both drum membranes and the ossicles were swept away following scarlet fever, the promontories are epidermatized and the head of the stapes buried in cicatricial tissue. Sound perception, however, is so good that she still has a positive Rinne and hears a whispered voice one foot away. She holds a secretarial position satisfactorily, and in conversation one would not recognize that she was deaf. Here we must suppose that a soft tone impulse emanating from the whispered voice and partially diffused through the atmosphere is then projected through a narrow passage, when it is again diffused over the whole promontory with sufficient force to convey impulses to the head of the stapes buried in cicatricial tissue, and the tone be sufficiently defined to be perceived by the organ of Corti. However absurd this may appear, if it were not so the whole theory of the physical propagation of sound impulses by molecular waves is untenable. The varying acuteness of sound perception in all deaf people is familiar to all otologists, as well as the influence of fatigue, atmospheric conditions and lowered bodily resistance. Fortunately, all these cases can be easily explained on other grounds than physical obstruction.

Otosclerosis still remains an unsolved problem in otology, so far as etiology is concerned. Fortunately, there seems to be no clinical evidence to show that it is any more prevalent than in the days of Politzer, who found very few cases that could be confirmed by autopsy. While there is no typical hearing test the triad of Bezold still remains the guide for a diagnosis.

The foregoing outline represents the gradual change that was taking place in our conception of the perception and conduction of sound impulses by molecular waves. In a previous paper we outlined the change that must take place in the otologists' point of view to conform to the new theory of sound resulting from electronic vibrations and its impulses propagated by electricity. This at once draws a sharp line of demarcation between the function of the perception and the conduction apparatus as for-

merly understood. Recently Crile has succeeded in isolating a human cell, demonstrating its polarity and an electric current.

In attempting to understand what happens when perception is being gradually lost we must analyze the cause, propagation and reception of sound impulses. Science has accepted the view that the cause of sound is due to electronic vibrations. These vibrations are transmitted to receptors, which are numerous in all the tissues of the body, each receptor being composed of many electrons and protons with a negative and positive pole generating electricity (Crile). These impulses having reached the receptors, are transmitted to the end organ by nerves having a special function, such specialized nerves always responding in the same way to any irritation, whatever the cause (Crile). For example, electronic vibrations coming within the sound area pass through the external canal to the receptor, where these impulses are propagated by electricity (Raymond) to the filaments of the auditory nerve, through which they pass to the end organ. Now, any damage to the receptors or to the cable transmitting the message must interfere with sound perception. Each receptor is analogous to an electric battery.

With this conception of the transmission of sound impulses we can understand how perception might be impaired by either a mechanical, chemical or bacterial cause. Among the mechanical causes otologists have always recognized boilermaker's deafness, due to the continuous loud percussion sounds, whose influence we can understand might have a damaging effect on the receptor similar to the running down of a battery.

Any chemical damage to perception would occur through the blood stream, as occasionally happens from lead, arsenic and tobacco. This bad result is more often seen in the optic and motor nerve than in the auditory. Taking up the last cause, we are familiar with the involvement of the auditory nerve as the result of an acute infection, and as the toxemia or bacteremia clears up perception is partially restored. If the bacteremia from some local process in the upper respiratory tract is active enough to overcome the protection of the lymph node, any subsequent exacerbation of the local process would continue to further damage the organ originally involved, for it is characteristic of such processes

that when a remote organ is involved any subsequent exacerbation resulting from that type of infection adds to the original damage in the same location. Instances are to be seen in the involvement of special groups of muscles, the endocardium, glomeruli of the kidney, gall bladder, appendix, etc. The special local process and its action in impairing sound perception has been given in previous papers.

The color sense has been built up through centuries of experience. Three or four thousand years ago only two colors were recognized and for years four were thought to be the limit. Today there are still cases of color blindness. Electronic vibrations, which gave us first red and then all the colors of the spectrum up to white light, are transmitted to the retina, where they pass to the receptor, meeting the delicate filaments of the optic nerve, through which they are propagated to the end organ. This analysis of the cause, transmission and reception of sound, color and light shows the analogy between the function of the organ of sight and the organ of hearing. This is further strengthened by a similarity in the structure of the organ of Corti and the retina.

It was difficult to understand how delicate sound impulses could be depended upon to reach the organ of Corti through a damaged and imperfectly functionating conduction apparatus. Under the new concept this is more readily understood. The basilar and tectorial membranes, the rods of Corti and the filaments of the auditory nerve make up a nerve mechanism whose relation to the propagation and reception of sound impulses by means of electricity is more in accord with clinical experience, for a specific nerve responds to stimulation to transmit those impulses for which it was intended. In the case of the auditory nerve we can understand pitch, rhythm and harmony as the result of electronic vibrations propagated by electricity and acting on an end organ designed to analyze sound impulses.

The significance of tinnitus at the beginning and throughout the course of slowly progressive deafness is also more clear. From the foregoing facts we shall have to conclude that the conduction apparatus, so-called, is simply a protective mechanism and that physical obstruction has nothing to do with the etiology or progress of the loss in tone perception, its only influence being

to retard in some measure the passage of electronic vibrations to the receptor. Whether the auditory nerve receptor is injured by loud sounds or as the result of a chronic infection, the final condition is nerve deafness.

The foregoing review of the changing attitude of otologists shows that any obstruction in the eustachian tube or middle ear could have but little effect in preventing the passage of sound impulses by electricity and no influence in effecting a further loss in tone perception. One of the strongest arguments in support of any theory is that it answers to all conditions and is not disturbed by any new discoveries in science. The writer has operated on hundreds of cases, over sixty years of age, for a chronic focal infection in the upper respiratory tract, where tone perception was being lost, and published hearing tests showing improvement in the hearing. This improvement lasted from six months to five years and was finally lost by a recurring exacerbation of the original local infection process. The baneful influence of such a chronic focal infection on remote organs was pointed out by Grünvalt, twenty years ago, and confirmed by Billings, Rosenow and many others. The brilliant work of Rosenow has been repeated so often at Rochester that W. J. Mayo remarked to the writer that it had ceased to be a matter of curiosity to him. This work has never been discredited by anyone following Rosenow's technic (Haden). On the other hand, Dr. Haden, who was at the head of the department of experimental medicine at Kansas University, after spending five years in the study of Rosenow's claims, was able to duplicate them all. The bacteriology, pathology and clinical histories of his cases, published about four years ago, are models of painstaking work. Such local chronic foci, especially in the tonsil, explain the deafness that accompanies a so-called catarrhal adhesive middle ear deafness or the loss of tone perception in any case. The new discovery that electronic vibrations cause sound impulses that are propagated by electricity presupposes the end result in the progressive loss of tone perception to be nerve deafness.

520 Commonwealth Ave.

LX.

THE CONDUCTION OF AIR SOUNDS TO THE COCHLEA.

A. G. Pohlman, M. D.,*

St. Louis.

The problem of the physics involved in the conduction of air sounds to the cochlea is by no means a modern one, and the history of the various interpretations is well worth a brief consideration. While the older observers at the time of the Renaissance possessed none of our modern facilities for investigation, they compensated in part for this deficiency through close analytical study. We, of the present day, may wonder how certain seemingly obvious facts were quite completely overlooked, and, on the other hand, we may also be impressed with a correct solution based on experimental evidence in spite of the crude and inadequate methods of the times. Thus Capavacci, in 1603, quite correctly established a differential diagnosis between a conduction and a perception deafness by comparing the acuity for hearing a zither through air with that experienced when a rod held between the teeth was applied to the body of the instrument. This method, however, merely put into practice the observation on bone transmitted sounds by Cardanus in 1560.

The discovery of the ossicles, by Berengario, in 1521, and by Ingrassio, in 1546, led to a functional interpretation suggested by Koiter, in 1575, that the drum membrane vibrations were conducted through the ossicular chain to the cochlea. It was probably Koiter's description which furnished the source for the first popular account of the mechanism of audition by Phineas Fletcher in 1633.

*From the Department of Anatomy, St. Louis University School of Medicine.

"As when a stone, troubling the quiet waters,
Prints in the angry stream a wrinkle, round
Which soon another and another scatters
Till all the lake with circles now is crown'd,
All so the aire struck with some violence nigh,
Begets a world of circles in the skie;
All which infected move with sounding qualitie
These at Auditus' palace soon arriving,
Enter the gate and strike the warning drumme;
To these three instruments fit motion giving,
Which every voice discern; then that third room
Sharpens each sound and quick conveys it thence;
Till by the flying poast 'tis hurried hence,
And in an instant brought unto the judging sense."

It must be remembered that at this time the internal ear was supposed to be filled with air. Indeed, if one looks at the proposition quite without prejudice, was there any reason for the older investigators to suspect that the perceptive mechanism for air sounds would be located in any other medium than that conducting the sounds to the ear? For that matter, how many of the modern investigators have considered the seemingly anomalous condition of an end organ of exquisite sensitivity developed as an immersion receptor? Had the internal ear really been filled with air, as the skeletal preparations seem to indicate it was, the condition might have been quite reasonably interpreted as an ideal environment for the sound analyzing mechanism.

Schelhammer, in 1684, demonstrated that the auditory tube is functionally closed, so far as the conduction of air sounds is concerned, by observing a vibrating fork held in the mouth. He differed with the accepted idea that the sound conduction took place through the ossicles and believed that drum membrane vibrations were conducted through the air of the middle ear to affect the cochlea by way of the round window. This interpretation might have been a perfectly proper one had the internal ear been filled with air. It was not until Cotugna, in 1774, demonstrated the liquid nature of the contents of the bony labyrinth that the accepted idea of an air filler was corrected. Scarpa, in 1789, separated the bony from the membranous labyrinth and believed that air sounds might be conducted through both windows.

The next outstanding contribution, following the work of Astley Cooper on the drum membrane, was that of the physicist

Wollaston, in 1820. This observer found that the sensitivity for low frequency sounds was more seriously affected during the aspiration of the air of the middle ear through negative Valsalva than that for the high frequencies. This finding apparently led to the interpretation that the disability of the conduction apparatus results in a greater loss in sensitivity at the low frequency end of the audible range, and made it necessary to assume a second pathway for the conduction of the high frequencies. This carries the problem to the present time, where the accepted doctrine of sound conduction is that low frequencies are conducted through the ossicular chain while high frequencies pass directly through the air of the tympanic cavity and into the cochlea through the round window.

In an extensive and detailed symposium on audition before the German Otological Society meeting in Basel, in 1930, Professor Alexander of Vienna presented a paper on this particular topic of air conduction. Inasmuch as Professor Alexander is a recognized authority on otologic matters, it follows that his opinion is well worth a serious consideration, and the more so because the summary of his conclusions has been presented in a comprehensive and concise manner. The writer has taken the liberty of translating these conclusions to bring the discussion into an accessible form, and where a difference in opinion occurs there the evidence for the objection will be stated briefly.

Die Theorie der Luftleitung mit Berücksichtigung der Anatomie und Klinik. G. Alexander, Monatsch. f. Ohrenheilkunde, Vol. 65, 1930, pp. 1-20 and 173 to 200.

The problem before us is quite clear because the vibrations of the drum can be conducted to the cochlea through only two important routes, and with four major possibilities:

a. Conduction of all frequencies through the ossicular chain (Koiter, 1575).

b. Conduction of all frequencies through the air of the tympanum by way of the round window (Schelhammer, 1684).

c. Conduction of all frequencies through the both windows (Scarpa, 1789).

d. Conduction of the low frequencies through the ossicular chain and the high frequencies directly through the round window (Wollaston ? 1820).

The accepted interpretation today is the last named—i. e., a simultaneous dual conduction where low frequencies pass through the ossicular chain to the oval window and where high frequencies are conducted directly through the membrana tympani secundaria. It is this accepted interpretation which is championed by Alexander, and it is this interpretation which is opposed by the writer.

In statement No. 38 of the conclusions, Alexander issues a warning against the misinterpretation of the available data, which perhaps might have more properly headed his summary.

Inasmuch as it is one of the few statements with which the writer agrees, it may be considered an excellent starting point for this review.

"No. 38. We must avoid overpowering the facts. Our theory must place us in a position to readily explain all of the facts derived through anatomic study and through clinical observation. The application of facts, correct in themselves, toward passing judgment on theories of hearing, has been subject to repeated error. Comparative anatomic data has been mistakenly correlated with the physiology of the human ear. Further, today as opposed to past times, the premise has been accepted without convincing basis that only one method of tympanic air conduction could obtain, and without considering the possibility under conditions of pathologic changes in the ear, that a conduction of air sounds might occur which differs from that of the normal. Finally, clinical observations, capable of more than one interpretation, have been arbitrarily, and by no means convincingly, directed toward the explanation of the one or the other theory of hearing."

It must be noted that definite experimental evidence on the only form available for such study, the human being, is necessarily limited. It was for this reason that the earlier writers were forced to speculate, even as the present day observers have been driven to conjectural evidence. While conjectural evidence is quite permissible, and indeed often unavoidable, such evidence

must not be construed as definite proof. The question, therefore, is not on the merit of Professor Alexander's opinion, or even on my own, but rather, do the explanations proposed fit the facts as determined by the best scientific methods available at the present time? Surely, in the face of the statistical evidence on auditory disabilities, at least that part of the apparatus which is accessible to direct physical investigation, should be determined in such a manner that there can be no doubt on its physical responses. If, therefore, by reason of our discussion an interest is awakened in the problem of sound conduction that it may be properly explained, it may come to pass that both Professor Alexander and myself are quite wrong, just as it will be evident in this review that both cannot be correct.

Where the discussion of several statements may be made together, they will be grouped, although the reference number will be preserved. The writer has taken care to express the ideas as set forth by Alexander as he has stated them, because an attempt is being made to bring the discussion up to date and in an accessible form.

It is no more than fair, before a critical review of Professor Alexander's summary is attempted, that some attention be devoted to his analysis of the objections to conduction pathways.

"Objections to the Ossiculo-Cochlear Conduction."

"Anatomical-physiological facts.

"1. The various segments of the drum membrane conduct the sound pulses in a variably efficient manner. As opposed to this, the ossicular apparatus and drum membrane constitute a very delicate system for pressure increases—i. e., for pressure variations in the internal ear (Weinland-Beyer)."

Just as the effect on pressure variations in the air of the middle ear is a composite of all positional changes in the drum membrane, so the movements in the ossicular chain may be said to be a resultant of all forces acting. If then the ossicular chain shows evidences of a delicate response in the pressure variations in the internal ear, it may also be considered as an excellent conduction system for sound vibrations.

"2. The incus does not follow the lateral displacements of the malleus (Weinland)."

This was known to Helmholtz, but the idea of a displacement of the malleus from the incus, even if it might be possible in large movements of the drum membrane, in some forms, probably does not occur in any forms as a result of definite sound vibrational responses. The subjective observations by Helmholtz in listening to loud sounds may be interpreted on another basis than the one which suggested itself to him.

"3. The ossicular chain is not capable of rapid movements (Weinland)."

This statement might perhaps be more definitely stated that the ossicular chain does not appear capable of rapid motions in the form of a lever.

"4. According to Beyer, the drum membrane is not adapted to conduct as vibrations the many complicated tones as found, for example, in a concert. As opposed to this, Gutzmann compares the drum membrane and ossicular chain with a recording phonograph which is capable of reproducing concert music."

The reviewer also disagrees with Beyer, because the sound vibrations in the air of the middle ear are absolutely dependent on what is conducted through the drum membrane. Accordingly, if the drum membrane is poorly adapted to conduct the complicated vibrations, a distortion of the vibratory responses, both in the air of the middle ear and in the ossicular chain, would result.

"5. The drum membrane is practically inelastic."

The meaning of this statement hinges on the meaning of the word "elastic," which is commonly associated with the usual word "elastic," meaning the property to stretch. However, in physics, elasticity is related to the property of restoration after deformation.

"6. The pathway through the Fenestra vestibuli into the cochlea is indirect. The plane of the stapes footplate is about at 80° to that of the plane of the opening into the Scala vestibuli. Sound pulses of small amplitude would necessarily lose through diffraction, particularly those which must pass through the longest route."

Liquid pressures, however, are transmitted in all directions equally, and the small size of the cochlea in relation to the wave lengths of audible sounds makes this consideration of no great importance.

"7. The Scala vestibuli is narrow at the vestibule and becomes higher and wider through the depression of the Lamina spiralis toward the body of the cochlea."

Here again this relation may have little effect on the transmission of the pressures.

"8. Variations are found at the stapes and also in the folds of mucous membrane about the ossicular chain."

"9. Variations in the form and the position of the promontorium, of the round window and of the membrana tympani secundaria are also found, but no mucous membrane folds occur at the membrana tympani secundaria."

This seems to prove only that small variations have no particularly significant effects on the acuity of hearing, and therefore, on the efficiency for sound conduction.

"Facts from comparative anatomy."

"10. In Rana, the aquaeductus suffices for pressure compensations."

This statement was perhaps included because in the frog the round window is not related to the tympanic cavity. It may also be noted that the ossiculocochlear conduction is probably not well adapted in the frog, and how much a frog may really hear is, after all, rather indefinite.

"11. The course of the carotis externa through the stapes."

This will be considered later in the summary.

"12. Many vertebrates have acute hearing but no drum membrane."

This statement cannot be supported by the experimental evidence, as shown later.

"13. According to Beyer, the drum membrane in the bird is poorly adapted for sound conduction."

It is difficult to see how Beyer arrived at this conclusion, because if the drum membrane is poorly adapted to conduct sound, then the bird must be hard of hearing, irrespective of

whether the ossiculocochlear or the aerocochlear route is the conducting pathway.

"Clinical Observations."

"14. Excellent acuity after loss of the drum membrane, the ossicles or both."

This statement is denied by the writer in his consideration of the summary.

"15. Transient increase in acuity in otosclerosis after operative opening of the lateral bony semicircular canal."

This may also be due to freeing the path for the direct impact of air sounds and has also been found in such cases on perforation of the drum membrane. If the operation thins the bony wall, a slight additional increased acuity might obtain, but probably not one which would lie far beyond an experimental error.

"16. Influence on hearing and on subjective internal ear noises brought about through spinal puncture."

The explanation for this may not be directly related to sound conduction, since the mechanics of the middle ear remain unchanged.

"Objections to the aerocochlear conduction."

"1. Variations in the promontorium and in the membrana tympani secundaria are frequent in the human being"; and without particular effect on the acuity of hearing.

"2. Nuvoli holds that the membrana tympani secundaria is too small and could therefore only conduct a small part of the sound pulses. Stefanini opposed this because the membrana tympani secundaria in many mammals is very large."

Both of these statements, however, may be classed as conjectural evidence without an experimental confirmation.

"3. The scala vestibuli established a pathway embryologically before the scala tympani. The physiologically more important conduction pathway is completed first."

This may be correlation but it is not evidence.

"4. The anatomy of the reptilian ear, according to Beyer, is evidence against an aerocochlear conduction."

This again is conjectural evidence, and as will be shown later in the case of the human being, it is practically impossible to definitely separate the aerocochlear from direct bone conduction.

"5. Oppikofer found normal acuity in cases where the niche of the round window was completely filled with fat."

These cases were not tested audiometrically, and perhaps even then the variations in the acuity for hearing might not be different from the variable normal or the personal error in the observer, particularly at threshold stimuli.

"6. The method of the operation of the artificial drum membrane."

This is discussed later in the summary.

It will be noted that the objections against either the ossiculocochlear or the aerocochlear conduction do not include a definite consideration of the physical principles involved nor is any of the exact quantitative experimental work on the acuity in the normal and abnormal human being mentioned. Let us see in what manner Alexander develops the conclusions in his summary.

"No. 1. In all vertebrates, where an air containing middle ear is present and closed to the outside, and where the auditory tubes are also functionally closed, two conduction pathways for air sounds may be distinguished: an ossiculocochlear and an aerocochlear."

We may agree that vibrations of the drum membrane are conducted by the ossicular chain to the oval window and through the air of the tympanic cavity to the round window. However, it has been quantitatively demonstrated that the ossiculocochlear conduction is more efficient than the aerocochlear path by a factor of about 40 db. This means with normal perceptive apparatus that a relative intensity increase for a threshold stimulus through the aerocochlear route of about 10,000 times the normal is required. At this intensity level, however, the threshold for a cross-over effect is almost attained. Thus, given an individual completely deaf on one side with normal hearing on the other, if the telephone receiver is applied to the deaf ear, the normal ear will hear the signal by bone conduction when the intensity level of about 50 db. is reached. Accordingly, when an intensity in the air sounds comes to the point where it will produce a threshold stimulus by way of the aerocochlear route, one cannot completely rule out the vibrational responses in the medial tympanic wall as

a whole. For this reason a third route must also be considered in the direct conduction of air sounds through the medial tympanic wall, and indeed this was noted by Alexander in the substance of his paper.

"No. 2. The ossiculocochlear conduction passes by way of the drum membrane and ossicles to the oval window and through the scala vestibuli on through the helicotrema to the scala tympani."

This statement is similar to that of E. H. Weber (1851), who believed the labyrinth liquid was quite incompressible, and therefore described a shuttle action in the liquid of the two scalæ, with the round window membrane acting as a compensation opening. However, it must be remembered, if the labyrinth liquid is incompressible, then the pressure compensations at the round window must be synchronous with the displacements at the oval window. The shuttle action implies the two windows are quite rigidly connected and respond as if they were one structure. Thus if one takes a straight glass tube filled with water and provided with a membrane at either end the motions produced in the one membrane are in phase with those of the other. Merely bending the tube to the form of a U in no way alters the conditions because liquid pressures are transmitted in all directions. However, if the tube is bent into a U and pressures are applied equally to both windows, then the push on one side opposes the push on the other side, and a complete cancellation of all motions will result. One must avoid visualizing the relations and infer that the stapes footplate brings about displacements in the perilymph of the scala vestibuli which are propagated up this channel, pass through the helicotrema and then bring about displacements at the round window membrane. Such reactions are either instantaneous or are dependent on the speed of sound in water—i. e., practically instantaneous with the distances involved. It will also be noted that Alexander does not mention any reactions at the round window in reference to displacements at the oval window, and the reason for this will appear later. However, the motions brought about by sound are pendular motions with equal advance and retreat, and if a motion in the scala vestibuli results in a motion in the scala tympani then the reverse must also be true.

"No. 3. The aerocochlear conduction passes from the drum membrane through the air of the tympanic cavity to the round window and directly into the scala tympani by way of the round window membrane."

Here it will be seen that no mention is made of the displacements in the scala tympani passing through the helicotrema into the scala vestibuli. As was pointed out in the note on No. 2, if the plus phase in the scala vestibuli passes through the helicotrema into the scala tympani, then the minus phase in the scala tympani must result in a similar motion.

"No. 4. According to the anatomic relations in the cochlea, the physiologic stimulus to audition is brought about through the vibration of the basilar membrane. These vibrations result in positional changes in the ciliæ and are transformed by the auditory cells into terms of nerve stimuli distributed to the applied nerve terminals."

While the idea that the physical stimulus to the auditory cells may be brought about through vibrations of the basilar membrane is not entirely objectional, it must also be remembered that probably everything within the cochlea vibrates, including its walls. That the physiologic response in the auditory cells is caused directly through the displacement of the ciliæ may or may not be true, and at least has thus far not been demonstrated. In any event, the manner of the response in the auditory cells is not definitely related to the topic under discussion.

"No. 5. At rest the free ends of the ciliæ are in contact with the lower surface of the membrana tectoria."

This statement appears to be quite correct. However, it must not be inferred that when the end organ is not at rest the ciliæ are not in contact or even embedded in the tectorial membrane. It will be seen later that Alexander feels, under certain conditions, the tectoria may be out of contact with the ciliæ.

"No. 6. The interposition of the distensible but not elastic membrana vestibularis and the membrana tectoria between the liquid of the scala vestibuli and the organ of Corti results in a great intensity loss through this conduction pathway. Losses of this amount could be tolerated physiologically only in the conduction of low tones."

"No. 7. Under these conditions the aerocochlear conduction establishes a direct route, particularly for high frequencies, which leads to nerve stimulation without intensity loss."

"No. 8. It may be assumed that for the transmission of low tones, the movements in the perilymph which begin at the oval window and pass up the scala vestibuli become physiologically operative in producing basilar membrane vibration after they have passed through the helicotrema into the scala tympani."

"No. 19. If one agrees with the viewpoint that sound conduction as a physical quantity ends within the cochlea in the vibrational responses in the basilar membrane, then it clearly follows that sound conduction through the scala tympani is more effective than that through the scala vestibuli because of the direct application of the liquid in this scala to the basilar membrane. In the case of the scala vestibuli, the membrana vestibularis and membrana tectoria act as a barrier to the transmission of the displacements in this scala toward the basilar membrane."

"No. 20. It, therefore, becomes necessary to assume that sound pulses conducted through the ossicles to the scala vestibuli must pass through the helicotrema to the scala tympani to become physiologically effective at the lower surface of the basilar membrane."

It is at once evident that Professor Alexander is trying to make the available data conform with his idea that the basilar membrane is affected physiologically only by the displacements applied to its lower surface. The advice, as set forth in No. 38, has seemingly been disregarded, and, however logical the sequence, the premises are in direct violation of the physical principles involved.

Sound pulses are pendular motions, irrespective of the medium in which they are conducted. If the membrana vestibularis and membrana tectoria have a damping effect on the reactions of the basilar membrane to the pressure changes in the scala vestibuli they will have a similar effect on the responses from the scala tympani side. A damping factor applied to a vibrating membrane on one side affects the response, whether the vibrations are applied to the one or the other side. Accordingly, Alexander's

proposal to activate the basilar membrane only from the scala tympani aspect cannot be accepted. A similar objection may be made to the idea that we dismiss the physics with vibrational responses in the basilar membrane because a reaction of this sort can only come about with vibration of the medium applied to both sides. The statements numbered 6, 7, 8, 19 and 20 must be regarded as conjectural evidence which is not in harmony with the facts. A similar objection may be entered to the explanation that greater intensity losses are physiologically tolerated best at the low frequency end of the audible range because, in point of fact, this is the least sensitivity area, and therefore, objectively as well as subjectively, can stand an intensity loss in conduction least well.

"No. 9. Normally, with closed auditory tube, the ossiculocochlear and aerocochlear conduction pathways are simultaneously operative. The ossiculocochlear conduction is, however, of greater psychophysiologic importance in spite of the fact that with closed auditory tube the aerocochlear conduction offers the principal, if not the only route for high frequencies."

This statement must be discussed not only from the standpoint of the physics but also from that of the experimental evidence in normal and abnormal cases. A physicist would first define the terms low and high frequency and then ask, if the low frequencies pass through one window, what keeps the high frequencies out, and vice versa? The second question might be, Would sound vibrations in the intermediate frequency range pass through both windows? Let us define our terms and grant that 128 Hertz is a low frequency and 8192 Hertz is a high frequency. Then, according to Alexander, all, or practically all, of 128 Hertz enters the cochlear through the ossiculocochlear route, while all, or practically all, of 8192 Hertz passes through the round window. As the frequency rises one may assume that somewhere between these two frequencies a pitch level will be attained where the displacements are effective at both windows. If such displacement were simultaneous and of equal power then no motional reactions in the basilar membrane might occur at all. Accordingly, the sensitivity of the ear should fall as the frequency rises from 128 Hertz and the area of least sensitivity should be found

somewhere about 1024 Hertz. However, as first established by Wien, the reverse is true because the ear is most sensitive at the region 1024 to 2048 Hertz.

We are, therefore, driven back to the point of fact. Is it true that high frequencies, say, of 4096 to 8192 Hertz, are conducted efficiently through the round window or is this merely conjecture? The answer to this question is offered in the information derived on some 47 cases of stapes fixation. According to the accepted ideas, these individuals should hear the high frequency end of the audible range at normal threshold intensity and should show great loss in the acuity for low tones because the ossiculocochlear apparatus is functionally out of commission. In point of fact, these cases demonstrate about an equal loss in sensitivity for all tones between 64 and 8192 Hertz and require an intensity increase through the entire range by a factor of some 10,000 times the normal threshold stimulus in spite of the fact that the perception apparatus is quite normal. This may be interpreted to mean that all of these individuals heard all of the frequency range equally poorly through the aerocochlear conduction, and also that the ossiculocochlear conduction is about equally efficient in the transmission of all usual audiofrequencies.

It was mentioned in the introduction to this review that there was no reason why the older observers should have even suspected that the internal ear was filled with anything else than air. The physicist realizes that the resistance difference between air and water is so great that an air sound pulse is almost completely reflected back at the surface of the water. The physicist also knows that merely placing a membrane on the surface of the water to which the air sounds are applied does not change this condition materially, because then the membrane takes on characteristics dependent on the liquid applied to it. In this connection the following bit of experimental evidence is worth citing: Kleinschmidt (1901) listened to sounds conducted through the water by means of a funnel connected with the external auditory canal. He found that the intensity with which the sound was heard was not greater with a membrane placed across the opening of the funnel over that heard directly from the surface of the liquid.

The physical problem of getting the air sounds into the water is one of overcoming the resistance differences. In this connection the principle employed in phonographic recording may be stated. Here a large diaphragm flanked by air on both sides, with the air sounds applied to only one side, is connected to the resistant surface (wax) by means of a lever stylus. The function of the lever is to decrease the motion and increase the power. One might assume that this would be an ideal arrangement for the ear where a larger drum membrane is connected through the ossicles to the smaller stapes footplate applied to the perilymph. If now the ossicles acted as a lever the functional efficiency of the system would be increased. However, the idea that the ossicles do act as a lever, first proposed by Valsalva in 1704, is confirmed by neither the physical nor the experimental evidence. Further assuming the Dahmann explanation that the leverage action in the ossicles compensates for losses in the transmission and that a leverage action in ratio of 5-4 really obtains (which the writer does not grant), then the intensity increase due to lever action would be as 16 to 25, or an increase of 50 per cent. This would be insufficient to be of any physiologic consequence. It must also be remembered if a transformer system is essential to efficiently conduct sounds of low frequency from air to water then a transformer is also required at the high frequency end of the audible range.

The absolute test on the validity of this information may be determined only in the human being in which one of the conduction pathways (the ossiculocochlear) has been lost. Indeed, Alexander mentions this condition in the following two statements:

"No. 27. In middle ear defects after the loss of the drum membrane, the ossicles or both the aerocochlear conduction may become increasingly important, particularly in cases with closed auditory tubes. The most favorable condition, anatomically, at the membrana tympani secundaria, and therefore that of the greatest efficiency in aerocochlear conduction, is found in cases after radical mastoid operation in which the air acuity is excellent. Here in all cases, in spite of the otherwise excellent sensitivity, sounds up to about 50 Vd. are not heard by air conduction.

The ossiculocochlear conduction is absolutely essential for frequencies of this order."

"No. 42. The method of operation of the artificial drum membrane permits of a variable explanation, according to one's inclination, as evidence for the ossiculocochlear or aerocochlear conduction."

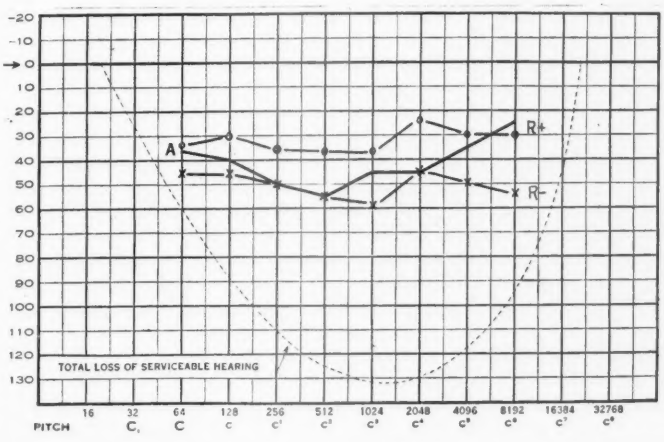
These two statements, as has been said, are definitely related to the experimental evidence in the human being. Alexander quotes from Weinland that one advantage in the artificial drum membrane may be due to a preventive of the rapid dissipation of sounds from the middle ear. This explanation, however, leaves something to be desired, because anything which will prevent air sounds from "escaping" from the middle ear at the same time also prevent their entering.

At the International Congress at Copenhagen, in 1928, the writer presented a brief review of the experimental work performed at the Riverbank Acoustic Laboratory. It was mentioned in this paper that the evidence pointed to a practically equal efficiency of the ossicular chain in conducting sounds of all usual audiofrequencies. The quantitative results on the effects of prosthesis in a case of bilateral radical mastoid, by Lillie and Kranz, and also the effects on the air acuity curves brought about by a bilateral ossiculectomy, were reported. This last named case experienced great benefit through application of a glycerin-cotton prosthesis and returned for examination some five years after the operation. She was tested on a Western Electric 2a audiometer on three different occasions, with and without the prosthesis, and the averaged information is submitted in the accompanying chart.

This type of audiometer delivers eight octave frequencies from 64 to 8192 Hertz, inclusive. The O line on the chart indicates the average normal air acuity, and depressions are represented in decibels, as shown at the left of the figure. An increase of 10 db. means an intensity increase of 10; 20 db. $= 10,^{2}$ or intensity increase of 100; 30 db. $=$ an intensity increase of $10,^{3}$ or 1000, etc.

The case without prosthesis was in a condition of complete loss of ossiculocochlear conduction. If the accepted interpretation of

a dual transmission is correct, she heard only through an aero-cochlear pathway and accordingly should show little or no disability so far as the high frequencies are concerned. It will be noted in following the x-x line that the acuity was about equally depressed throughout the entire range and she heard all frequencies exhibited equally poorly. This case can be made to conform with the accepted interpretation in only one way. If the individual was perception deaf at the high frequency end, then this perception deafness might have been overcome through more efficient conduction of the high frequencies through the round



The *x* line presents averaged sensitivity of three separate examinations without prosthesis and the *o* line the averaged sensitivity with prosthesis. The patient was Rinne negative under the former conditions and Rinne positive with prosthesis.

window. That this is not true is shown by comparing her acuity curve with the average acuity curve of stapes fixation cases in which there is a normal perception apparatus. It is further indicated by the fact that with prosthesis the enhancement to air acuity was by a factor of about 20 db. in the lower frequency range, while in the higher frequencies the improvement was even more pronounced. The end organ must accordingly be quite

normal. It may also be noted that with prosthesis the case was Rinne positive while without she was definitely Rinne negative.

The point that conduction deafness cases tend to show about equal disability at all frequency levels required some further explanation because then why should conduction deaf individuals experience greater losses in sensitivity for low tones than for high tones? If one looks at the chart again, one may observe a faint broken line which was established by Wegel as the intensity level at which total loss of serviceable hearing may be anticipated. It will be seen that the region of the greatest sensitivity (from 1024 to 2048 Hertz) is at the same time the area of greatest intensity range. Below 1024 Hertz and above 2048 Hertz the absolute sensitivity of the ear falls especially at the high frequency end. Now suppose a case of complete conduction deafness, with even loss in the air sensitivity through the entire range by a factor of about 40 db. This case will probably not hear 48 Hertz with normal perceptive apparatus nor will 16384 Hertz be heard. At 64 Hertz the sensitivity range remaining will be about 20 db.; at 1024 to 2048 Hertz, 90 db., and at 8192 Hertz 55 db. Accordingly, the greatest relative loss is found at the low frequency end, where the sensitivity is least pronounced and the relatively least loss will be experienced between 1024 and 2048 Hertz where the sensitivity is the greatest. This, however, has nothing to do with the efficiency of the sound conduction system but is dependent on the normal sensitivity, which varies with the frequency as a function of the normal perceptive apparatus, and obtains alike for ossiculocochlear, aerocochlear and direct bone transmission. The major premise upon which all of the dual conduction explanations have been predicated is accordingly not in harmony with the experimental evidence. This statement has a practical application because if a given individual hears any frequency at normal intensity, the normality of the conduction apparatus for all frequencies is at once established.

"No. 10. The route through the ossicular chain in normal individuals with closed tube is at least not the only route for the conduction of air sounds."

This statement is quite true but with limitations. In the presence of a functional ossiculocochlear transmission, the conduc-

tion of air sounds through the round window is so inefficient at all frequencies that it may be quite disregarded. All individuals with nonfunctional ossiculocochlear conduction are deafened by a factor of about 40 db., even with normal perception apparatus, and this represents about the maximum usual deafness which may be found due to conduction disability.

"No. 11. The membrana tympani secundaria is elastic and capable of vibration and is in a position to conduct sound pulses to the scala tympani."

This statement is quite true as limited above. Had the internal ear been filled with air, as Schelhammer believed, then his interpretation would have been a correct one. Inasmuch as the internal ear is filled with liquid the vibratory responses in the round window membrane are limited by the liquid applied to it.

"No. 22. The aerocochlear conduction is important for high frequencies and for the highest tones in normal human beings; for other frequencies it is not important."

It has been shown above that the aerocochlear conduction is equally unimportant at all frequency levels.

"No. 15. It must be remembered in every clinical theory of sound conduction that air sounds pass through the air of the middle ear to reach the cochlea. This observation has been established through tuning fork diagnosis."

This statement is quite true again in a limited sense, because all complete conduction deaf cases tend to show the Rinne negative response and audiometric methods indicate one cannot definitely separate the aerocochlear conduction from the direct bone conduction through the medial tympanic wall at the intensity level required for a threshold stimulus.

"No. 12. The complete capacity to a vibratory response in the membrana tympani secundaria is only found when the intracochlear pressure is slightly positive, at zero, or slightly negative. Under the last named conditions the membrana tympani secundaria in histologic sections is slightly concaved toward the middle ear."

If the membrane responds equally well under conditions of normal and of increased or decreased intralabyrinthine pressure,

it is also logical to assume that the condition of its convexity or concavity has nothing to do with its alleged function.

"No. 13. Under these conditions the aquaeductus cochleæ may act as a pressure compensation opening."

"No. 14. The aquaeductus and the lymphatic channels of the internal auditory canal also afford pressure compensation for the sound pulses conducted to the vestibule through the ossicular chain."

"No. 16. The cochlea and labyrinth are anatomically united into one organ because both serve for orientation and both are physiologically activated by the movements in the liquid contents of the internal ear. Hearing is brought about by movements in the perilymph, which arise through the sound pulses coming from without. Labyrinth stimulation results on account of passive movements in the endolymph, which are caused by passive or active displacements of the head or the entire body. The cochlea is the organum perilymphaticum, while the labyrinth is the organum endolymphaticum. The pressure compensation openings for acoustic displacements are found in the aquaeductus cochleæ and in the lymph channels of the internal auditory canal. The safety valve for the endolymph is found in the canalis reuniens and the ductus endolymphaticus."

These statements must be answered more or less at length. The vibratory responses in the membrana tympani secundaria have already been noted. Inasmuch as liquid pressures are transmitted equally in all directions, it follows that whatever compensation openings are present, they must be operative in terms of the entire liquid filler. The separation of an organum perilymphaticum from an organum endolymphaticum is not quite clear, because the basilar membrane is flanked by the endolymphatic cochlear duct on the one side and by the perilymphatic scala tympani on the other side. Vibratory responses in the membrane, therefore, affect both the endolymphatic and the perilymphatic systems, and, accordingly, any pressure regulation factor on one system must be effective for both. The anatomic reason why the cochlea is associated with the labyrinth is due to its development as an evagination from the sacculus.

"No. 21. The evidence for a physiologic pressure compensation by way of the aquaeductus cochleæ and the lymph channels of the internal auditory canal is found in the observation of early deafness which results from acoustic tumors as soon as the internal auditory canal is completely filled and where the aquaeductus is destroyed, even when the nervus cochlearis still manifests a partially intact fiber system."

This statement is probably quite true and requires no comment.

"No. 17. The topographic differences in the resulting liquid displacements (in the labyrinth that of the endolymph and in the cochlea that of the perilymph) are related to the perilymphatic septa of the vestibule in such a manner that in spite of the anatomic connection of the systems labyrinth stimulation never gives rise to audition nor does a sound stimulus cause the vestibular responses of vertigo or nystagmus."

This explanation does not appeal to the writer as entirely satisfactory because it has already been pointed out the two systems may not be considered as definitely separated in the manner described. It is not unlikely that the character of the stimulus in relation to the definite functional adaptations of the end organ may be responsible for the result. In any event this part of the discussion is not related to the topic.

"No. 18. Under normal conditions the auditory tube is functionally closed. This arrangement prevents sound pulses from striking the drum membrane on both sides, which would result in limiting the vibrational responses. Further, it prevents a disturbing autophonia and isolates the drum membrane from respiratory pressure changes."

Here Professor Alexander and the writer are quite in agreement, except under conditions of open tuba, according to his own interpretation the high frequencies might be heard even more acutely.

"No. 23. The appreciation of sound direction—i. e., spatial orientation of the individual by means of sound, is dependent on the ossiculocochlear conduction. It must be remembered, however, that in final analysis the judgment of sound direction is a cerebral function."

This statement is by no means self-explanatory. Alexander again quotes from Weinland's article. This writer believed that the directional hearing is brought about through the manner in which the ciliæ are displaced in reference to the tectorial membrane, and on whether all of the auditory cells or only part of them are in contact. This conjectural evidence for lack of contact of the cilia with the tectorial membrane is also employed to explain paracousis Willisii.

"In a noise, the tectorial membrane is pushed down farther so that the vibrations of the ciliæ attain a contact with the lower surface of the tectoria, which relation does not obtain under usual conditions in these cases. In this manner the hearing better in the presence of a noise may be explained."

It is quite generally accepted by American observers that hearing better in a noise is not a real phenomenon but is dependent on the masking effect of the noise on the normal individual speaking which causes him to increase the intensity of his voice.

If Professor Alexander means, in his statement that spatial recognition is dependent on the ossiculocochlear pathway because this route is far more efficient than that of the aerocochlear conduction, then no discussion is necessary. However, we cannot agree to the assumption that the direction of the ciliæ displacement in reference to the membrana tectoria is the factor which determines the ability of the individual to recognize the direction from which the sound comes. Suppose a sound comes from the north: according to this interpretation, then because the sound pulses are pendular motions the ciliæ must vibrate, say, north and south. Suppose the sound comes from the opposite direction, then the ciliæ also vibrate north and south. Accordingly, one could not differentiate the two and no orientation would be possible. Individuals with one normal and one deafened ear find great difficulty in locating sounds, and a similar result obtains in cases with both ears deafened. Some mistake is being made in attempting to correlate the functional responses of the vestibular apparatus with that which takes place in audition.

"No. 24. The viewpoint that only one route for air sound conduction is present—i. e., the idea of a uniformity of sound conduction—must be abandoned. Beyer, Zimmermann and others

have assumed this uniformity in air sound conduction on the basis of a biased evaluation of the comparative anatomic and clinical evidence which, however, is susceptible of more than one interpretation."

Alexander is referring to the possibility that the aerocochlear conduction is the only pathway for air sounds to the cochlea. He does not mention the other route, the ossiculocochlear, which the writer maintains is the important conduction for all usual audiofrequencies.

"No. 25. In the human being the ossiculocochlear and the aerocochlear conduction are synchronous functionally. The first named is the more important in normal individuals with closed auditory tubes. In pathologic cases, after loss of the drum membrane, the ossicles, or both, the aerocochlear pathway becomes the important one. The ossiculocochlear conduction is absolutely essential for the conduction of low tones to about 90 Vd."

This statement has already been explained. Naturally, if the ossiculocochlear conduction is gone, the only remaining pathway is an aerocochlear one, if indeed it may be absolutely separated from the direct bone transmission through the medial tympanic wall. The reason why the aerocochlear conduction raises the lower limit of tone range is entirely dependent on the inefficiency of the conduction pathway and the relative insensitivity of the internal ear at this frequency level, as previously explained.

"No. 26. In the condition of open tube and in normal individuals, at least at the time of tubal opening, low tones, with the possible exception of the lowest, are conducted to the cochlea through the aerocochlear pathway."

If the tube is open and the air sounds are transmitted through the nose, then the pressure developed in the middle ear is probably greater than when the sound is applied to the drum membrane. However, it must be remembered that with air sounds applied to both sides of the drum membrane a cancellation effect on the resulting vibrations takes place. This effect would probably be more pronounced at the low end of the tonal scale not only because here greater diffraction of sounds to the external ear will occur, but as Kranz and the writer have demonstrated, a small tube has a greater effect on the efficiency with which high

frequencies are conducted than at low frequencies. The reason why low tones are not heard through the aerocochlear route has already been explained.

"No. 28. If, during the healing after radical operation in cases which show an excellent hearing, acoustically detrimental scars are formed at the round window membrane then the efficiency of the aerocochlear conduction is decreased and results in a lowered acuity of hearing."

This statement may be quite correct because the thicker the membrana tympani secundaria and the thicker the mucous membrane covering the medial tympanic wall the greater the intensity probably required to activate the end organ. However, to class any case of radical mastoid as having excellent hearing is not quite fair, because all of these cases have a pronounced lowering in the air acuity for all frequencies, and all tend toward the Rinne negative, even if they are not definitely negative in this response.

"No. 29. On the other hand, if the secondary pathologic changes, for example, the formation of delicate ligaments between the deep surface of the membrana tympani secundaria and the wall of the scala (the normal relations in the dog), through which the tension and vibrational responses in the round window membrane are increased, then the functional importance of the aerocochlear conduction will be considerably greater."

This statement is conjectural evidence. It would not seem unlikely that if scars on the outer surface of the round window membrane decrease the efficiency of its response, then scars on the deep surface might have a similar effect. Even if the condition described as normal for the dog obtained it would still be necessary to prove that this had any definite effect on the acuity for high tones, one way or another.

"No. 30. The two windows may operate each as a conduction and as a compensation opening but not synchronously. A simultaneous response at both windows by a qualitatively similar tone is not possible. One may, however, assume that in the instance of qualitatively different tones both windows may respond at the same time—i. e., both act as conduction and compensation openings."

This statement is that of an opinion not supported by facts and is in seeming contradiction to No. 9. If the aqueductus cochleæ, the lymph channels of the internal auditory canal and the ductus endolymphaticus may operate as compensation openings, then there is no reason to suppose that both windows may not be simultaneously operative. Indeed this is demanded in the instance of qualitatively different tones to account for the dual conduction where the low frequencies are supposed to pass only through the oval and the high frequencies only through the round window membrane. The physics involved appears to indicate if the windows are operative simultaneously in the conduction of different frequencies, then they are also operative in the simultaneous conduction of all frequencies. In any event the burden of the proof rests with Professor Alexander to support his own assumption. The pressure experiment of Bezold indicates that both windows may be operative simultaneously.

"No. 31. Cases with pathologic occlusion of both windows show marked deafness, even when the organ of Corti is quite intact. If the cochlea itself is somewhat disabled, then the deafness may be quite complete."

The writer has had no experience with cases of this type and therefore can make no comment excepting that with a pathologic process involving both windows the end organ itself might be affected.

"No. 33. As the bulla increases in size, the round window comes to lie in close relation to it and the membrana tympani secundaria tends to face the bulla."

So far as the writer is aware this is a correct statement.

"No. 32. Vertebrates are not uniformly adapted in reference to air conduction because with rudimentation of the ossicles and with increase in size of the round window, the importance of the aerocochlear conduction becomes greater."

This evidence is purely conjectural and is not supported by experimental findings.

"No. 34. The increase in the size of the bulla in animals may be regarded as a criterion of an increasing efficiency in the aerocochlear conduction. In this respect, the cat presents the peak of the development because the middle ear region is almost separated

into a lower part which forms the bulla with the associated round window, and an upper which contains the ossicles."

Physically the pressure responses in the middle ear region are dependent on two important factors: the vibrational reactions of the drum membrane and the amount of air trapped in the closed middle ear cavity. If, therefore, the development of the bulla actually increases the amount of air contained in the middle ear, a decreased efficiency in the aerocochlear conduction will result which would undoubtedly affect the sensitivity for all frequencies. Alexander does not show the experimental evidence which favors his assumption, and it may be that the bulla has no function in reference to an intensity response.

"No. 35. Bulla and fenestra cochleæ are particularly well developed in those vertebrates which react strongly to very high tones and in those animals in which orientation is accomplished through the ear rather than through the eye and the nose."

The writer has thus far found no experimental evidence for this statement. The only forms which have been described as having a very accurate orientation through sound, and even here but incompletely investigated, are birds; the owl, for example, which has no bulla. This evidence again must be classed as purely conjectural.

"No. 36. With increase in the size of the membrana tympani secundaria in a number of mammals, particularly in rodents and in some carnivores, the membrana tympani secundaria gains attachment to the lower surface of the lamina spiralis ossea and this increases its elasticity and vibrational response."

It has been previously noted the conjectural evidence that it would reduce the efficiency is just as good if not better; and either way one must await experimental confirmation before such evidence may be accepted.

"No. 37. The dog, throughout life, exhibits a delicate perilymphatic network at the blind end of the scala tympani, which forms an indirect attachment between the membrana tympani secundaria and the lamina spiralis ossea. This appears to increase the vibrational capacity of the membrana tympani secundaria and therefore increase the efficiency of this membrane to conduct the sounds by the aerocochlear pathway."

This statement was considered before, and the evidence might be equally well employed toward a damping effect on the membrana tympani secundaria. At least, there is no experimental evidence on the absolute acuity in the dog for hearing high tones nor on the importance of this assumed connection so far as an aerocochlear pathway is concerned.

"No. 40. The fact that in many mammals the carotid artery passes through the stapes is regarded in the literature as evidence against the ossiculocochlear conduction. This, however, cannot be substantiated, because the artery in no way interferes with the movements of the stapes, and in most animals in which this occurs the artery is closed in a bony canal which is soundproof. This relation disturbs the stapes as little in its physiologic responses as does the close relation of the carotid artery in the carotid canal to the cochlea influence the functional responses in the latter."

The idea that the presence of the carotid artery at the stapes does not materially affect the ossiculocochlear conduction for small amplitudes is probably quite correct. However, the assumption that it may not affect the conduction of large amplitudes is not warranted nor is the comparison to the relations of the artery at the cochlea mechanically a comparable quantity.

"No. 41. The idea that the membrane of the round window lies in the sound shadow of the drum membrane does not obtain for many mammals at all, and only in part for the human being. In man the primary drum membrane is not placed at 90° to the secondary drum membrane."

Here Professor Alexander and the writer are in agreement. The small size of the middle ear cavity probably makes it act as an acoustic capsule with pressures quite evenly distributed over the entire limiting surface and quite irrespective of the positions of the two windows. Air pressures in the middle ear obviously affect both stapes footplate and round window.

"No. 43. Pathologic changes in the human middle ear are conceivable in which a decrease in the ossiculocochlear conduction is accompanied by an increase in the efficiency of the aerocochlear conduction to the point of surpassing that of the normal."

This is Professor Alexander's opinion, and my own is that the condition is not conceivable.

"No. 44. The aerocochlear conduction must be considered in the construction of the aids to hearing, particularly if an increased acuity is sought for those individuals with loss of the drum membrane, ossicular chain or both."

It has been demonstrated that a Rinne negative individual with intact drum membrane shows the same quantitative acuity curve as one with normal apparatus except he is about equally deafened throughout the usual audio range. It has also been shown that a Rinne negative case without drum membrane and ossicles has a similar curve to that of the stapes fixation cases. All types of conduction deafness, irrespective of the cause, unless the external canal is occluded or the drum membrane loaded, show the same disability, and all conduction deafened cases therefore require the same sort of acoustic aid. This, however, is not true for perception deafened individuals, because here an actual deformation in reception is found, for which no aid to hearing has been devised better than cupping the hand back of the auricle because of a selective effect on high frequencies.

In will be seen in this review that an obvious difference in opinion is found between Professor Alexander's analysis of the problem of air conduction to the cochlea and my own. The attempt has been made to show that the simultaneous dual conduction with pitch limitations, which is the accepted explanation and championed by him, cannot be accepted. It may also be noted that both in the objections to the dual conduction interpretation, and in support of the functional ossiculocochlear conduction at all frequencies, effort has been made to avoid conjectural evidence so far as it is possible. The work of Weinland, extensively quoted by Alexander, has not been accepted by investigators of the physics or the physiology of the auditory apparatus in so far as I am aware. The extensive study by Beyer on the comparative anatomy of the vertebrates is undoubtedly biased by his opinion that the aerocochlear pathway must be the only efficient route for the transmission of air sounds. It is unfortunate that Alexander did not include in his review the extensive audiometric investigations notably by Bunch, Drury, Fowler, Jones and Shambaugh, nor was the equally important work on the physics of audition by such men as Fletcher, Kranz, Knudsen

and Wegel mentioned. To this must be added the researches by Quix, Struycken and Zwaardemaker of Holland, and more recently of Dahmann, Ruf and Sonntag in Germany. All of these investigations are definitely related to the problem of air sound conduction and none of them may be disregarded.

It would hardly be in keeping with the spirit of this review if the writer did not express his own opinion in a concise manner similar to that of Alexander. Perhaps the following statements will suffice:

1. The cochlear duct is developed from the older labyrinth specifically by an extension of the sacculus.

2. This results in the production of an immersion end organ which is relatively deaf to air sounds but affords the perception apparatus a place of reasonable physiologic quiet.

3. The appearance of auditory end organ is accompanied by a translation of the environment from an aquatic to a terrestrial habit and suppression of gill respiration.

4. The cephalic end of the gill apparatus becomes modified into a drum membrane and solid sound conduction system (ossicles or columella).

5. The function of the drum membrane and ossicular chain (columella) is to act as a transformer, which tends to match the resistance differences between the air and the liquid of the internal ear.

6. The larger drum membrane is accordingly flanked with air on both sides and with sound vibrations applied to one side only on account of the functional closure of the auditory tube.

7. The relatively larger drum membrane is directly connected to the smaller area of the stapes footplate through the sound conduction apparatus.

8. A leverage action of the ossicles in transmitting the drum membrane vibrations to the liquid does not appear to be mechanically probable nor is the amount of intensity increase which might be even theoretically possible of any great physiologic consequence.

9. The sound vibrations are conducted to the cochlea through both the ossicle chain and through the air of the tympanic cavity.

10. The former route is, however, so much more efficient than the latter at all usual audio frequencies that the aerocochlear conduction may be completely disregarded in the presence of a functional sound conduction system.

11. Where the ossicular chain is structurally or functionally lost, there the aerocochlear transmission becomes consciously operative, although the deafness through this route is by a factor of about 40 db., or an intensity increase of about 10,000 times the normal threshold stimulus at all frequency levels.

12. The aerocochlear conduction cannot be definitely separated from direct vibration of the entire medial tympanic wall and perhaps should not even be separated from this route.

13. The development of the scalæ in relation to the cochlea follows the rule for the development of the remainder of the perilymphatic spaces. The auditory end organ developed in the form of a strip and was accompanied by the increase in the size of the cochlear ganglion which likewise took on the form of a ribbon. This resulted in the production of a double septum in the cochlear. The scala vestibuli arose in relation to the upper septum of the cochlear duct or membrana vestibularis, and the scala tympani was applied to the other septum or basilar membrane. These two scalæ became confluent at the only point where such confluence is normally possible: the distal or tip end of the cochlear duct.

14. The position of the two windows in relation to the two scalæ and to the basilar membrane suggests that pressure applied equally to the two windows may have a cancellation effect on the movements of the basilar membrane. This might be interpreted as an automatic shock absorber which is instantaneously effective and therefore an ideal protection to the end organ against excessive motions. In any event this seems to be in accord with the experimental evidence by Bezold.

15. A transformer is required to conduct air sounds efficiently to the cochlea and no case with conduction deafness, or where the ossicles are removed, can be said to have excellent hearing acuity for air sounds. The bone acuity may, however, be quite normal.

16. There is no experimental evidence which favors a pitch limitation in the conduction of sounds to the cochlea, whether by the ossiculocochlear, the aerocochlear or by direct bone transmission.

17. The accepted interpretation of a dual conduction of air sounds to the cochlea appears to have been erroneously predicated on a pitch-limited efficiency in the conduction systems. The subjective findings, however, are quite successfully explained on the basis of the normal sensitivity as a function of the frequency in the perceptive mechanism. It is this fact which is brought to consciousness through conduction deafness, and which has been misdirected in its application to the problem of air conduction.

Accordingly, the individual who registers Rinne negative at one frequency tends to respond in a similar manner at all frequencies. If he does not, the difference may be attributed to the relative efficiency of the various forks in delivering air and bone sounds. Similarly, in making comparative tests at higher frequencies, the older examiner must have a care that he does not include his own degree of presbyacousia in his interpetation of the information obtained. This is true, because the greater the degree of presbyacousia in the examiner, the more pronounced the apparent deformation in the sensitivity range of his conduction-deafness patient.

LXI.

AUDITORY TESTS IN CASES OF MARKED OR COMPLETE UNILATERAL DEAFNESS.*

C. C. Bunch, Ph. D.,†

St. Louis.

The problem of standardization of the functional tests of hearing has been the "bête noire" of the otologist. Committee after committee, both national and international, have endeavored to bring order out of chaos, but a critical glance through the current journals reveals almost as great confusion today as existed at the beginning of the present century.

Audiometry has since its advent been subject to the keenest scrutiny by otologists. Has audiometry solved the problem of standardization? Does the information secured by audiometric methods add to that which could be obtained by methods already in vogue? Are audiometric tests practical from the standpoint of time and expense? Are the results secured capable of clinical interpretaion? Are they actually factual? These questions can be answered only by time and experience, and pitfalls of technic and interpretation should be made known.

One of the earliest criticisms to audiometric methods arose because in using the telephone receiver it is brought in contact with the head and offers the possibility for the transfer of energy to the perceptive apparatus through the medium of the cranial bones rather than through the air alone. The German investigators have apparently decided against the telephone receiver in favor of the loud speaker, but the necessity of stimulating each ear separately has caused the American investigators to prefer the receiver in spite of this criticism.

The statement has been made by some writers that because of this transmission by direct contact between the telephone receiver

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and the head, it is impossible to eliminate the perception of the ear not being tested and therefore the results secured by audiometric tests are false and lead to false conclusions.

Various methods have been devised to eliminate the effects of one ear when the other is being tested, especially when there is a marked difference in the acuity of the two ears. These methods usually take the form of the simple closure of the external canal, the use of a stream of air or water in the canal or the production of a noise. The last method has been called masking. Bárány devised his Larem apparatus for this purpose and it has been rather widely used by later otologists. Some object to the use of this Larem since by its mechanical structure it is not possible to confine the sound entirely to the ear which should be masked and therefore, to a certain extent, it masks the ear which is being tested. Moreover, the sound which it produces is so loud and disagreeable to the patient that it tends to distract his attention. But scientifically the chief objection to the Bárány Larem is that no two produce sounds of the same pitch or loudness, and they, therefore, cannot be standardized. Since the use of a sound for masking seemed the most simple for this study, other types of masking apparatus were sought.

One type which has been described is in the form of a telephone receiver connected in an oscillating electric current, preferably a current which produces very impure rather than a pure tone. One of the simplest forms consists of a telephone receiver connected through an ordinary door bell transformer, with variable voltage steps, to an ordinary alternating current lighting circuit. The current, while being rated for electric appliances at, say sixty cycles, will give a tone far from pure. The voltage steps of the transformer will provide variable steps of loudness, for it seems almost axiomatic that one should not use louder masking sounds than necessary.

A commercial buzzer which also serves admirably for this purpose is the Western Electric 3A buzzer audiometer. This produces a very complex buzzing noise and is provided with an intensity regulating dial similar to the dials used on the other Western Electric audiometers.

The records presented in this study were made by using one of the latter type of buzzers. The tests were not made in a soundproof room for the reason that noises were used in masking and the effects of the soundproofing would by the nature of the experiment be vitiated. Had the tests been conducted in such a room, one change would have been noted, viz., the records of the normal ears without masking would have a slightly lower threshold —i. e., would have been slightly higher on the charts. The masking noise used, however, was sufficiently loud to more than overcome any accessory noises entering the room in which the tests were conducted. The tests were made by the writer or under his supervision, for most part on patients at the Johns Hopkins Hospital in pursuance of the program of the Otological Research Laboratory, and are presented here with the permission of those in charge.

In order to test the claim that a masking noise will, to a large extent, eliminate the hearing in the opposite ear, a series of normal observers were tested with the 1A Western Electric audiometer both with and without the opposite ear masked. The telephone receiver from the 3A audiometer used for masking was held in close contact with the ear not being tested by an ordinary spring headband. A soft rubber ear cushion was used to more closely confine the sound energy to the ear which was being masked. With the buzz from the 3A continuously sounding in this receiver the tests were conducted on the opposite side in the routine manner.

Fig. 1 shows the results secured from two of these observers. In all four charts the records indicated by joined circles, give the acuity for the ear being tested without the masking noise sounding in the opposite ear. The records of connected crosses show the acuity when the masking receiver at the opposite ear made a noise from the 3A audiometer with a loudness of 65 units. The latter in each case shows a threshold slightly lower, usually about five units, than the record of circles, although there are points where the two coincide. At other points the record of crosses is above the record of circles, indicating better hearing when a masking sound is in the opposite ear. This is probably accounted for by variations in the attention of the listener. The

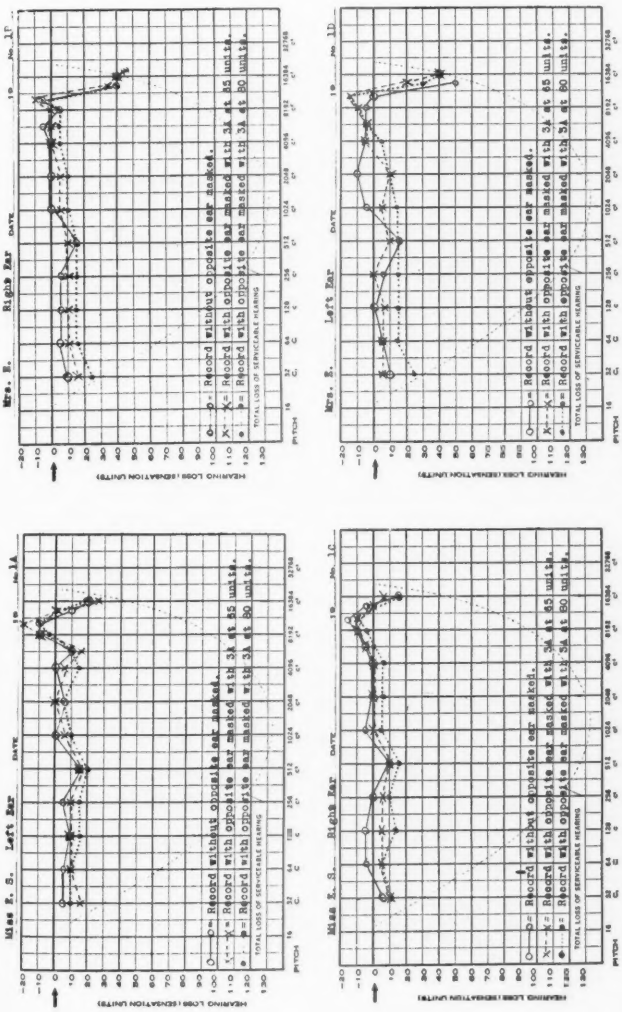
Mrs. E. Right Ear
Record without opposite ear masked.
Record with opposite ear masked with 3A at 85 units.
Record with opposite ear masked with 3A at 80 units.
Mrs. E. Left Ear
Record without opposite ear masked.
Record with opposite ear masked with 3A at 85 units.
Record with opposite ear masked with 3A at 80 units.
Miss E. S. Left Ear
Record without opposite ear masked.
Record with opposite ear masked with 3A at 85 units.
Record with opposite ear masked with 3A at 80 units.
Miss E. S. Right Ear
Record without opposite ear masked.
Record with opposite ear masked with 3A at 85 units.
Record with opposite ear masked with 3A at 80 units.
HEARING LOSS (SENSATION UNITS)
PITCH
TOTAL LOSS OF SERVICEABLE HEARING

Fig. 1. Audiograms showing the effects of masking the opposite ear in normal cases.

difference between the two records is for the most part not more than five units, a difference which may be expected in two successive tests on any patient when masking is not employed.

The records of connected dots were taken in each instance with the opposite ear masked with the noise from the 3A Western Electric audiometer at 80 units. It will be seen that these thresholds are distinctly lower at almost all points than the record of joined circles. It is true that there are points where all three curves coincide, but the curves of connected dots are for the most part from 10 to 15 units lower on the audiogram chart than the curves of connected circles. This indicates that a masking noise from the 3A audiometer of 80 units loudness applied at one ear while the audiometer test is being made will, in cases with normal hearing, cause the threshold of the opposite ear to be lowered from 10 to 15 units.

Fig. 2 is the record of a patient with complete unilateral deafness and with rather marked loss in acuity for high tones in the hearing ear. In this case it was desired to know to what extent masking the deaf ear in the manner above described would affect the thresholds of the hearing ear. The curve of joined circles indicates the acuity of the hearing ear when no masking noise was used. That of joined crosses indicates the

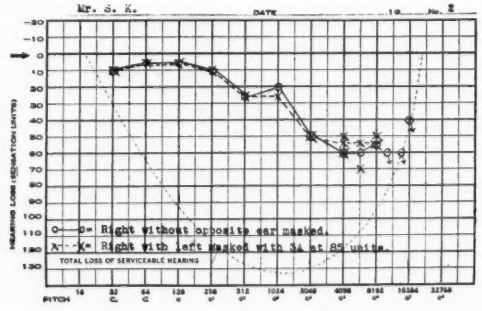
Mr. S. K.
DATE
No. 2
HEARING LOSS (DEVIATION UNITS)
O—O= Right without opposite ear masked.
X--X= Right with left masked with 3A at 80 units.
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

Fig. 2. Audiogram of a case with complete unilateral deafness. The record shows the effects on the thresholds of the hearing ear when a complex noise is sounding continuously in a telephone receiver at the deaf ear.

acuity when the deaf ear was masked with the noise from the 3A buzzer with 85 units of loudness. In this case the differences between the two records is very slight, and any variation in the two records can be accounted for by fluctuations in the attention of the listener. The masking noise at the deaf ear in this case had no measurable effect on the acuity of the hearing ear.

Fig. 3 shows the records of four cases of rather marked unilateral deafness. In Fig. 3A the difference between the thresholds of the two ears is sufficiently great that it might be suspected that the indicated hearing of the right ear is nothing more than that which might be perceived through and around the head by the normal left ear. However, when the normal left ear is masked with a noise from the 3A audiometer 65 units loud, the threshold of the right deafened ear is not lowered, as shown by the curve of joined dots. To satisfy ourselves that this amount of masking actually eliminated the normal ear, still louder masking was used at the normal ear, viz., the 3A at 85 units, and the curve of joined dashes secured. In this case, at least, masking the normal ear with a noise of 65 units or 85 units did not eliminate the residuum of hearing in the opposite deafened ear. Figs. 3B and 3C are records of similar cases where the results are similar when a masking noise of 65 units is used. In Fig. 3D the masking noise employed was 75 units. In each of these, by masking the normal ear with a very loud Bárány Larem, it could be demonstrated that the deafened ear was sufficiently acute to perceive spoken voice close to the ear, nor was there any definite reason for suspecting complete deafness.

Fig. 4 shows the audiograms of four cases of marked unilateral deafness with curves having different contour from those shown in Fig. 3. In Fig. 4A both right and left ears have markedly lowered thresholds, but the left is much less acute than the right. When the right was masked with a noise of 75 units from the 3A buzzer, the curve of joined heavy dots was secured. The curve secured without masking the better ear and that secured when masking was employed are within five units of each other for all tones, and when a noise of 90 units was used for masking, the change in the thresholds was within the same limit for all tones except one (512 d. v.).

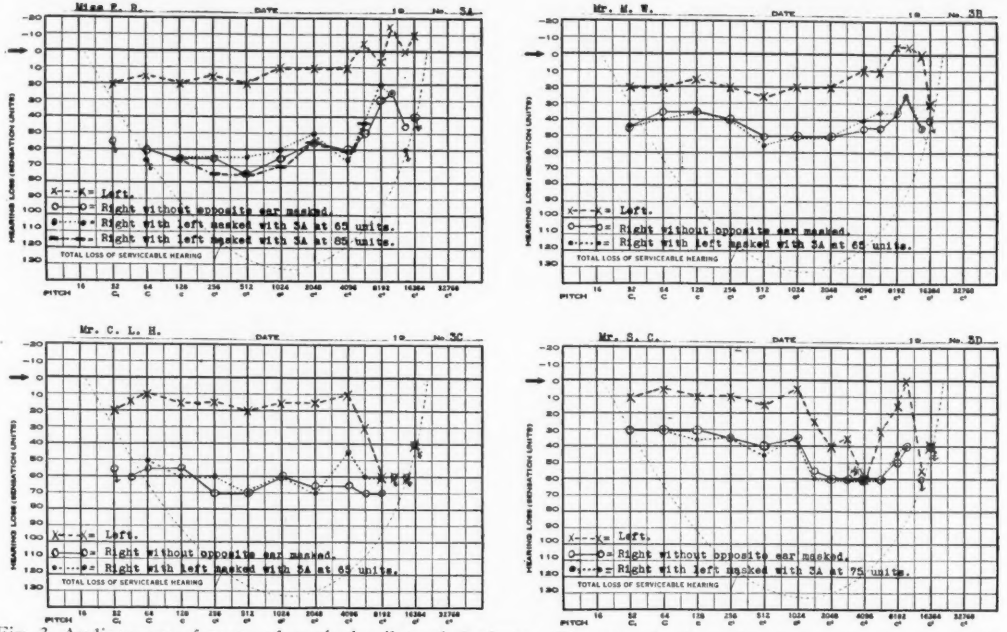
Miss P. B.
Mr. M. W.
Mr. C. L. H.
Mr. S. C.
X---X = Left.
O—O = Right without opposite ear masked.
Right with left masked with 3A at 65 units.
Right with left masked with 3A at 85 units.
Right with left masked with 3A at 75 units.
TOTAL LOSS OF SERVICEABLE HEARING
HEARING LOSS (SENSATION UNITS)
PITCH

Fig. 3. Audiograms of cases of marked unilateral deafness. The records show the changes in the thresholds of the deafened ears when a noise is sounding continuously in a telephone receiver at the better hearing ear.

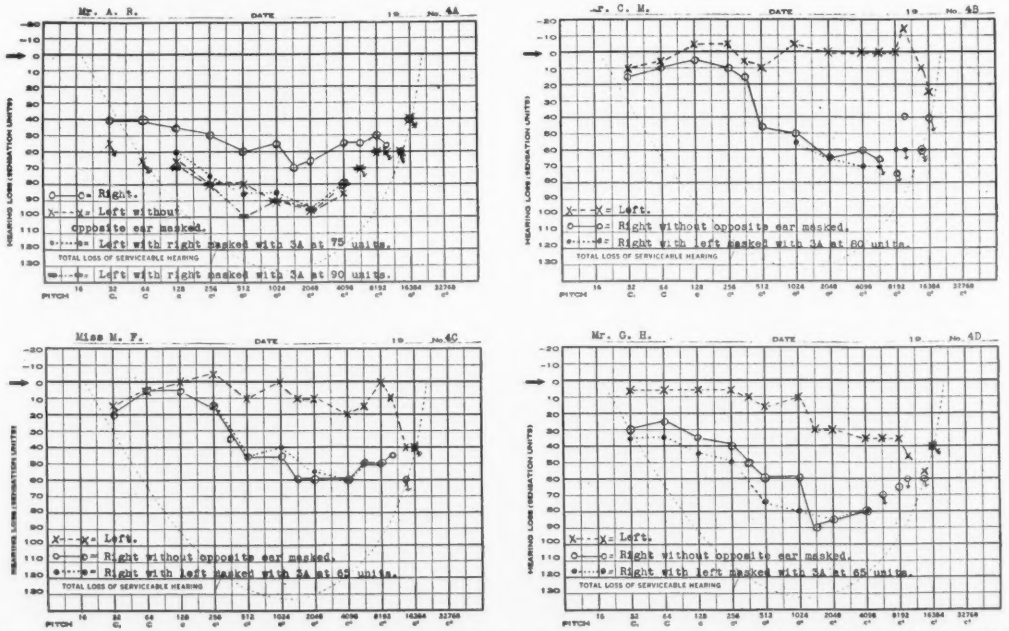
Mr. A. R.
DATE
No. 4A
o= Right.
X--x= Left without opposite ear masked.
o·····o= Left with right masked with 3A at 75 units.
o-·-·-o= Left with right masked with 3A at 90 units.
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
F. C. M.
No. 4B
X--X= Left.
o——o= Right without opposite ear masked.
o·····o= Right with left masked with 3A at 80 units.
Miss M. F.
No. 4C
X--x= Left.
o——o= Right without opposite ear masked.
o·····o= Right with left masked with 3A at 65 units.
Mr. C. H.
No. 4D
X--X= Left.
o——o= Right without opposite ear masked.
o·····o= Right with left masked with 3A at 65 units.

Fig. 4. Audiograms of cases of marked unilateral deafness. The records show the changes in the thresholds of the deafened ears when a noise is sounding continuously in a telephone receiver at the better hearing ear.

Fig. 4B shows the record of a case where the low tones are heard almost equally well in both ears, but there is a marked loss for high tones in the right. Only the high tones need therefore be masked. Masking the left ear with a noise of 80 units caused a slight lowering of the thresholds for the high tones in the deafened ear. This may be due to the interference of the better ear, but if one may judge by the evidence of the previous record it is improbable.

Fig. 4C is a record somewhat similar, where masking the better ear with 65 units caused no lowering of the threshold in the opposite ear.

Fig. 4D shows that masking with 65 units caused a slight change in the threshold for low tones, but that for high tones was unchanged.

Fig. 5 illustrates the records from three cases with total unilateral deafness. Their clinical histories were such that unilateral deafness seemed certain. In each record, if masking is not used, the totally deaf ear gives a curve which lies almost parallel to and from 30 to 60 units below that of the hearing ear. It is this type of case which has caused so much discussion in audiometric methods.

When a masking noise of 75 units is used at the right ear in Fig. 5A, no tones were audible in the left at the maximum intensity produced by the audiometer nor could the patient tell by the sensation of touch when the oscillating current was turned on or off in the receiver at the left ear. No hearing could be demonstrated in this ear by any method of testing available in the laboratory. The arrows in each case indicate the maximum intensity of the octave tones. The acuity for the semi-octaves was also tested but no responses could be obtained. Fig. 5B is similar to 5A except that the contour of the curve for the hearing ear is different.

It was suspected that the record for the left ear in 5C was in reality the amount which was heard through or around the head, so a masking noise of 65 units was applied to the right and it was found that responses only for tones between 256 d. v. and 1024 d. v. could be secured. This is shown by the curve of connected dashes. But when the masking noise in the right ear was

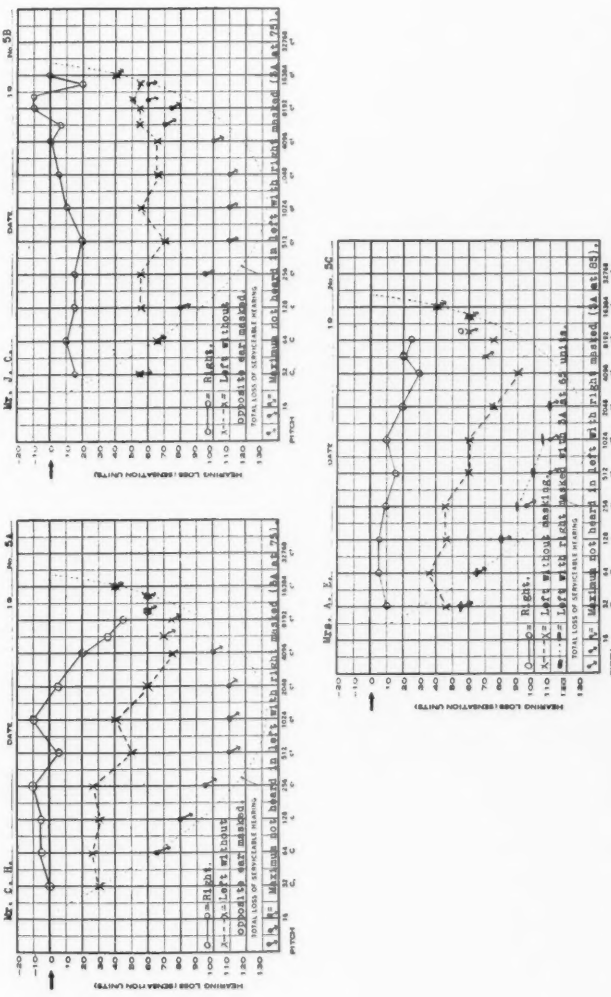

Fig. 5. Shadow curves, i. e., records given by totally deaf ears with and without the hearing ear masked.

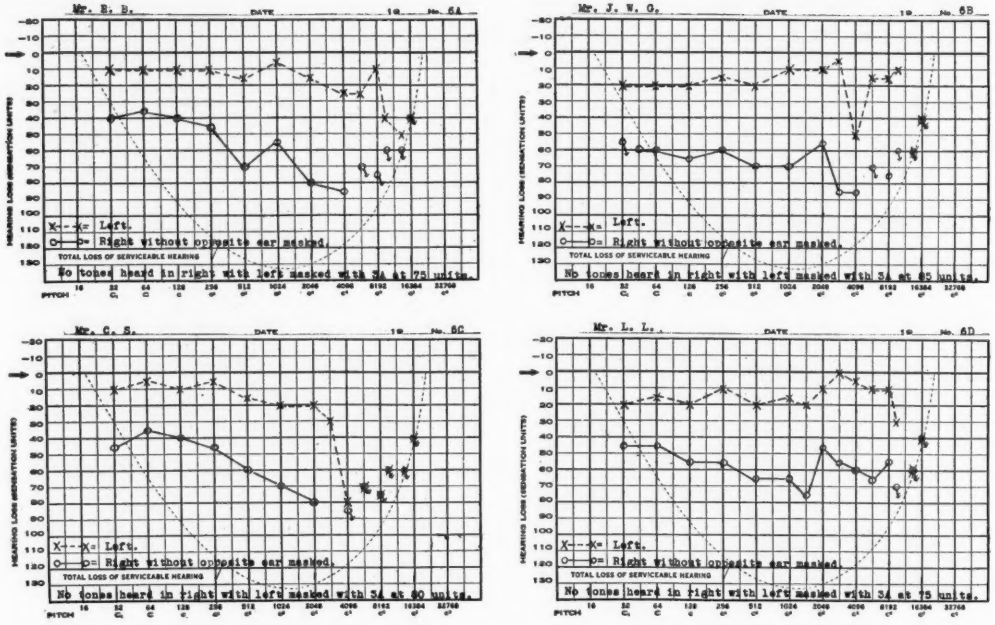
Mr. B. B.
No. 6A
Mr. J. W. O.
No. 6B
Mr. C. S.
No. 6C
Mr. L. L.
No. 6D
X--X= Left.
O--O= Right without opposite ear masked.
TOTAL LOSS OF SERVICEABLE HEARING
No tones heard in right with left masked with 3A at 75 units.
No tones heard in right with left masked with 3A at 85 units.
No tones heard in right with left masked with 3A at 90 units.
No tones heard in right with left masked with 3A at 75 units.
PITCH

Fig. 6. Shadow curves, i. e., records given by totally deaf ears with and without the hearing ear masked.

increased to 85 units no tones at the maximum intensity of the audiometer could be heard in the left.

The term "shadow curve" has been applied to describe this type of record. Since these ears are known to be totally deaf, the hearing indicated by the curve for the deafened ear in each case is in reality a "shadow" of the hearing in the opposite ear.

Fig. 6 shows the shadow curves secured from four other cases of total unilateral deafness. When a record is obtained in which the curves for the two ears lies parallel to each other and thirty or forty units apart, one should immediately suspect that the indicated hearing of the poorer ear is in reality a shadow of the hearing in the better ear and should, by some method of masking, attempt to eliminate the possibility of the acuity of the better hearing ear entering into consideration when the thresholds for the poorer ear are being determined. In Figs. 6A and 6D masking noises of 75 units were sufficiently loud. A noise of 80 units was used in the case whose record is shown in Fig. 6C, and one of 85 units was used in 6B; but with the case whose record was shown in chart No. 5C a noise of 65 units was not sufficient.

The record shown for the right ear in chart 7A was thought to be a shadow curve. However, when a masking noise of 75 units was used at the left, had this been a true shadow curve, no curve should have been obtained if the evidence secured from the cases illustrated in Figs. 5 and 6 hold true. But with a masking of 75 units at the good ear, a curve was obtained in the deafened ear which was entirely different in contour from that secured without masking. Fig. 7C is somewhat similar to 7A. The case shown in 7B was extremely interesting, for when the right ear was masked only one tone (91 d. v.) was perceived out of the entire twenty-two produced by the 1A audiometer. It does not seem probable that this reaction was due to the sensation of feeling, since no response could be secured from tones a half octave higher or lower in pitch.

Fig. 8 illustrates further possibility of demonstrating by this method the presence of islands of residual hearing, if they may be called such. When a masking of 80 units was applied to the good ear in the case whose record is shown in Fig. 8A, an island extending from 91 d. v. to 256 d. v. was shown; 342 d. v. was not

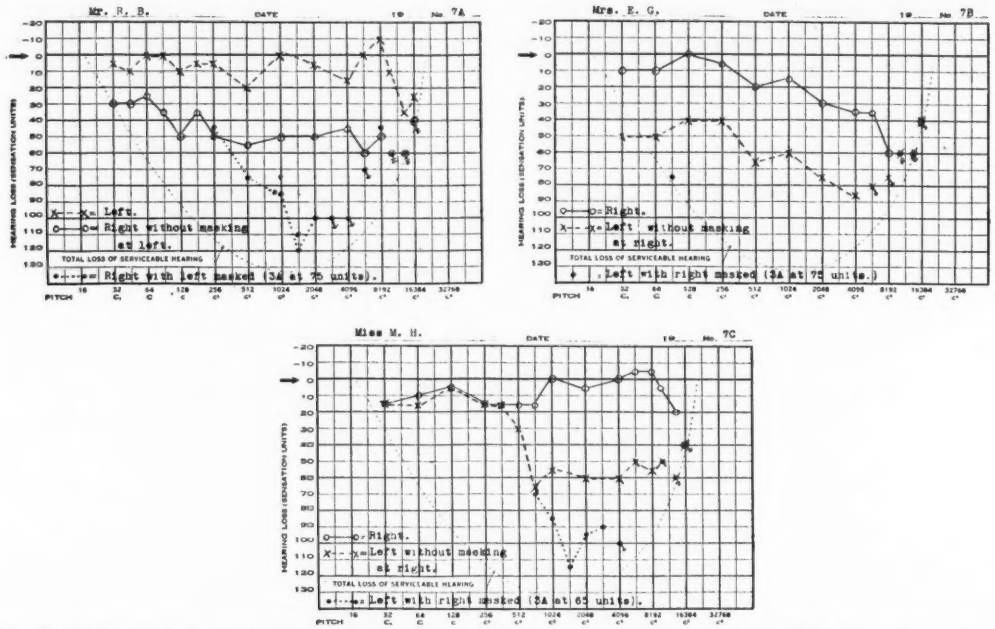
Mr. R. B. DATE 19 No 7A
HEARING LOSS (SENSATION UNITS)
X—X= Left.
O—O= Right without masking at left.
TOTAL LOSS OF SERVICEABLE HEARING
•----•= Right with left masked (3A at 75 units).
PITCH
Mrs. E. G. DATE 19 No 7B
O—O=Right.
X—X=Left without masking at right.
TOTAL LOSS OF SERVICEABLE HEARING
•----•=Left with right masked (3A at 75 units.)
PITCH
Miss M. H. DATE 19 No 7C
O—O=Right.
X—X=Left without masking at right.
TOTAL LOSS OF SERVICEABLE HEARING
•----•=Left with right masked (3A at 65 units).
PITCH

Fig. 7. Audiograms of cases with marked unilateral deafness in which curves of different contour were secured when the opposite ear was masked.

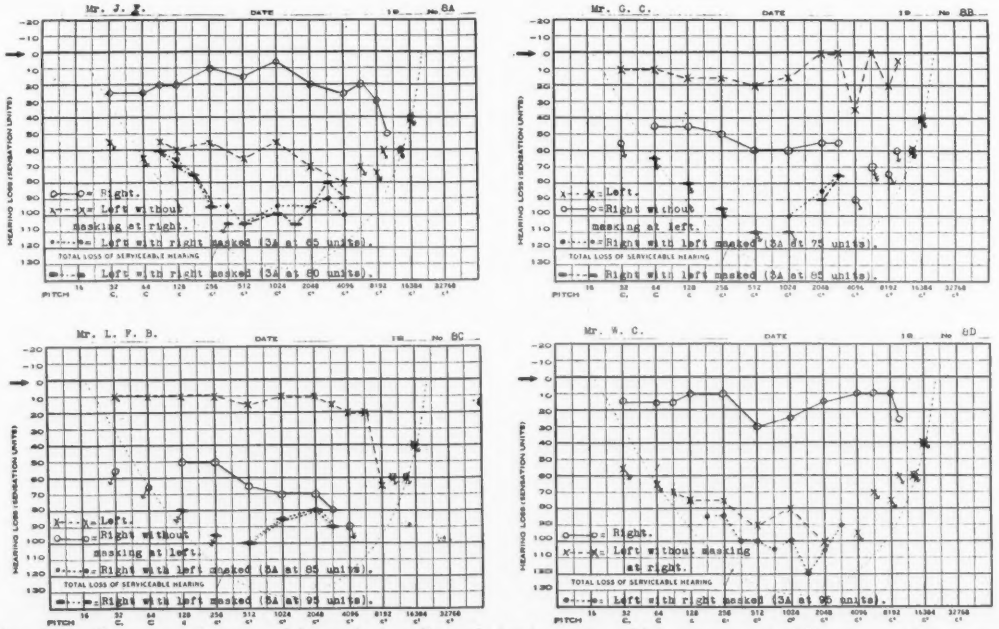
Mr. J. F.
DATE
No 8A
HEARING LOSS (SENSATION UNITS)
o—o = Right.
x- - -x = Left without masking at right.
•----• = Left with right masked (3A at 65 units).
TOTAL LOSS OF SERVICEABLE HEARING
= Left with right masked (3A at 80 units).
PITCH
Mr. G. C.
DATE
No 8B
x- - -x = Left.
o—o = Right without masking at left.
•----• = Right with left masked (3A at 75 units).
= Right with left masked (3A at 85 units).
Mr. L. F. B.
DATE
No 8C
x- - -x = Left.
o—o = Right without masking at left.
•----• = Right with left masked (3A at 85 units).
= Right with left masked (3A at 95 units).
Mr. W. C.
DATE
No 8D
o—o = Right.
x- - -x = Left without masking at right.
•----• = Left with right masked (3A at 95 units).

Fig. 8. Audiograms of cases with marked unilateral deafness in which curves of different contour were secured when the opposite ear was masked.

heard at its maximum, and tones from 512 d. v. to 4096 d. v. were heard. Fig. 8B shows an island in the right from 2048 d. v. to 2896 d. v. when the left was masked with a noise coming from the 3A buzzer at 85 units. The island in Fig. 8C extends from 512 d. v. to 2896 d. v. That in 8D extends from 181 d. v. to 2896 d. v.

These may not be true tonal islands. It is possible and quite probable that by increasing the intensity of our stimulating tone these islands may merge to form continents, so to speak. The Western Electric 1A audiometer is the loudest source of relatively pure sound which has been devised for testing purposes.

The question immediately arises as to the necessity and practicability of this method of masking. Certainly in all cases, in practice, diagnosis or research, if it is essential to know the presence, location and extent of the elements of residual hearing, the method is the most exact which has been devised, to the best of the writer's knowledge.

CONCLUSION.

No attempt has been made to prove that masking sounds applied to the good ear in cases of unilateral deafness do not affect the hearing in the deafened ear. It has, in fact, been shown that such masking does affect the opposite ear, at least in normal cases. It has also been shown that by the proper selection of masking apparatus and with proper intensity control it is possible to prevent the hearing of the good ear from entering into consideration when audiometric tests are being done in cases of marked unilateral deafness.

LXII.

EFFECT OF SUPPURATIVE DISEASE OF THE TEMPORAL BONE ON CERTAIN OF THE CRANIAL NERVES.*

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Certain cranial nerves may be affected directly or indirectly by suppurative disease of the temporal bone. That a cranial nerve is affected may be assumed to indicate that intracranial extension has taken place from the temporal bone. The problem of accurate anatomic and pathologic diagnosis is made more exacting under such circumstances. Stereoscopic visualization of the anatomic relation of the parts demonstrates that a lesion causing the symptoms need not be extensive. The lesion affecting the nerve may be due to congestion, to localized inflammation of the dura, to localized abscess formation, to diffuse serous or suppurative meningitis, or to none of these causes. The lesion is most often situated in the petrous pyramid. The clinical picture as a whole must be given careful consideration. The fact that a cranial nerve is involved is not, in itself, I believe, sufficient evidence on which to institute radical surgical measures.

During the last few years many interesting articles have appeared in the literature which have dealt with the theoretic and practical application of present knowledge of the pathologic processes involving the petrous pyramid.

Eagleton stated that the apex resembles the metaphysis of the long bones in structure and in function, that it continues to grow until late in life and that it may be "unlocked" by surgical measures. The apex is a marrow-containing bone; it is the exception to find pneumatic cells in it. Infection produces a peculiar type of osseous suppuration, local nutritional death with marked sur-

*From the Section on Otolaryngology and Rhinology, The Mayo Clinic. Read before the American Otological Society, Briarcliff Manor, New York, June 18 to 20, 1931.

rounding protective reaction, or the infection may be limited to periostitis of the apex.

Kopetzky and Almour expressed the belief that there is a period between the mastoid lesion and the terminal lesion in the meninges resulting from the infection in the petrous pyramid in which the patient is almost free of symptoms. This has been suggested from roentgenologic studies of the petrous pyramid. Their explanation of the mechanics of the invasion of the apex is based on the Wittmack theory of pneumatization. The petrous pyramid may be affected by two varieties of suppuration, frank suppuration and osteomyelitis. In all of their cases well pneumatized petrous pyramids were present. Profant has suggested the name petrositis for this disease.

Peet stated that he would rely much more on homolateral dilation of the pupil than on paralysis of the sixth nerve as a diagnostic sign. Ray reported a case of paralysis of the sixth nerve on the side opposite to the lesion of the mastoid. Mollison reported two cases of paralysis of cranial nerves in association with disease of the mastoid, the third nerve in one, and the sixth nerve in the other. In the latter there was an increase in the pressure of the cerebrospinal fluid. He said: "It is probable that some form of meningitis was responsible for the involvement of individual cranial nerves, although the evidence is poor." Watson-Williams reported finding at necropsy a fibrinopurulent mass extending across to the opposite side and surrounding the third, fourth and sixth nerves on both sides, and the fifth, seventh and eighth nerves on one side, in a case of left facial paralysis associated with disease of the right mastoid. The spinal fluid was cloudy. Thrombosis of the right side had extended to the occipital sinus.

Lyman reported paralysis of the fifth and seventh, and a portion of the third nerves following a radical operation on the mastoid. Lillie and Lillie, and Jesberg reported choked discs without intradural involvement. Robertson reported a case with thrombosis of the sinuses, paralysis of the sixth nerve and choked discs.

Meltzer stated that if one does not agree that the existing suppuration is actually the cause of the lesion in the majority of

cases, one can readily fall in line with the theory of inflammatory edema at the tip. Friesner and Druss brought out clearly that there are several routes by which infection or the effects of infection may reach the apex. Shambaugh, in discussing Friesner and Druss' paper, said that the conclusion that surgical interference is indicated should not be reached hastily because of the paralysis of the sixth nerve. Eagleton and Kopetzky and Almour recently described a clinical syndrome of suppuration of the petrous pyramid.

Disease of the pyramid may explain involvement of the fifth, sixth, seventh and eighth cranial nerves, but it does not account for chocked discs, neuralgia of the branches of the fifth nerve or of the jugular foramen syndrome.

MATERIAL STUDIED.

An interesting series of cases has been encountered in which certain of the cranial nerves were found to be affected in association with both the acute and chronic types of suppurative otitis media and mastoiditis. Naturally, it was presumed that the problem of diagnosis and treatment was more exacting. Of the cranial nerves, the second, fifth, sixth, seventh, eighth, ninth, tenth and eleventh have been found involved either independently or in groups in association with suppurative otitis media and mastoiditis.

Second Nerve.—In approximately 10 per cent of the cases of thrombosis of the lateral sinus, changes are visible in the discs. However, choked discs may be associated with disease of the mastoid without thrombosis of the sinuses or intradural extension of infection. Four such cases were reported in 1925, and three others have been observed since then. The mechanism of choked discs in such cases cannot be easily explained, although usually the condition is ascribed to disturbance in the sulcus lateralis. One patient was found to have homonymous hemianopsia of the congenital type, associated with surgical mastoiditis. Patients with congenital homonymous hemianopsia are not cognizant of the visual defect until it is called to their attention; the acquired type, however, is very troublesome. All the patients in this group recovered.

Fifth Nerve.—In one case definite neuralgia of the mandibular branch of the left trifacial nerve was a perplexing symptom associated with mastoiditis. The patient was a boy who had had an operation on the mastoid two weeks previously. He came to the clinic because the pain had not been relieved. Sharp, severe pain started in the region of the first lower molar tooth, extended to the temporal region and disappeared after a few minutes. The pain recurred several times a day. The facies was an index of the severity of the pain. The tooth had been removed without relief. A second operation on the mastoid revealed the dura over Trautman's triangle to be very red, the zygomatic cells and the cells along the posterior wall of the canal were extensively involved, and a small sequestrum was present on the posterior wall of the canal. Following the operation, the pain was completely relieved. Kopetzky stated that this phenomenon may be explained by irritation of the small superficial petrosal nerve through the otic ganglion, as the mandibular branch is connected with the otic ganglion. The irritation may occur in the tympanic plexus or along the course of the nerve.

Fifth and Sixth Nerves.—The fifth nerve, or more usually the gasserian ganglion, may be involved along with the sixth nerve in so-called Gradenigo's syndrome. Eleven such cases have been observed. The pain varies in severity; if it is very severe, spinal drainage may afford great relief. Only two of the patients in this group were subjected to operation on the mastoid; both operations revealed involvement of the cellulæ petrosæ. All of the patients recovered. Two patients were found to have definite changes in the spinal fluid; one patient presented the classic clinical picture of serous meningitis. The cells in the spinal fluid were too numerous to count. Repeated drainage relieved the patient completely. Three years later he returned with paralysis of the sixth nerve on the opposite side and with reinfection of the ear. Exploration of the mastoid was undertaken with disappointing results. The petrous pyramid was diploic in type and seemed not to be involved. The symptoms were not severe at any time. After two months the paralysis disappeared. Recently a little girl was referred to the clinic because of paralysis of the external rectus muscle associated with otitis media. The history revealed

that typical serous meningitis had occurred with spontaneous recovery. She may have been in the interval of which Kopetzky spoke. There seemed to be nothing to do in the way of treatment.

Seventh Nerve.—Recovery may take place in cases of paralysis of the seventh nerve and acute otitis media. If evidence of mastoiditis is definite, surgical interference is indicated. The fact that the seventh nerve is paralyzed does not constitute a real indication for operation unless the condition of the mastoid warrants operation. I have observed three such cases. In one case the seventh nerve was found to lie in an anomalous position in the petro-apical groove—in fact, it was lying free. The function of the nerve recovered almost completely. The seventh nerve is more frequently affected in chronic suppurative otitis media because of the nature of the disease. I believe, therefore, that under such conditions surgical interference is indicated. Twelve such cases have been observed.

Eighth Nerve.—The vestibular branch of the eighth nerve may be affected in acute otitis media either because of inflammatory irritation or by pressure on the windows. Operation on the mastoid relieves the symptoms if myringotomy does not. Four such cases have been encountered. In the chronic form of otitis media twenty such cases have been encountered. The labyrinthine symptoms disappear quickly or gradually. Definite disease of the labyrinth in this connection will not be considered here for obvious reasons.

Seventh and Eighth Nerves.—There is, however, a syndrome involving the seventh and eighth nerves which may be explained on the basis of an inflammatory lesion at the cerebellopontine angle, which I have observed in three cases. The vestibular reactions showed that the canals on the same side did not function. The symptoms of dural irritation and serous meningitis were alarming, and surgical interference was instituted because it was felt that the infection had extended through the labyrinth to the meninges, as there were definite changes in the spinal fluid. The Neuman operation was performed. The labyrinth was found to be normal apparently, but the dura over the apex was very red, and it was felt that the seventh and eighth nerves could

have been involved in the internal auditory meatus. One patient had a fistula in the horizontal canal, but the other canals and the vestibule seemed not to be involved. All the patients recovered, although the seventh nerve did not regain function.

Ninth Nerve.—The glossopharyngeal nerve was found to be affected in one case, associated with chronic mastoiditis, but removal of an infected tooth relieved the symptoms referable to the nerve.

Ninth, Tenth and Eleventh Nerves.—These nerves (the jugular foramen syndrome) were involved following operation on the mastoid in one case. Later a lateral pharyngeal abscess formed; it was drained and the symptoms disappeared. It is apparent that this was an extradural abscess. Eagleton has given an explanation for this phenomenon by stating that the disease is directly below the periosteum and does not involve the medulla.

SUMMARY.

Fifty-nine cases were observed in which there was involvement of cranial nerves, either independently or in combination, with suppuration of the mastoid process. The natural inference under such circumstances is that the disease of the mastoid is of a more serious nature than usual. The fact that a cranial nerve is involved is of unusual significance, and, therefore, the clinical picture as a whole must be carefully considered in order that well directed treatment may be instituted.

THE MAYO CLINIC.

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LXIII.

CONGENITAL STENOSIS OF THE LARYNX: REPORT OF THREE CASES.*

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The literature of congenital stenosis of the larynx is scant; textbooks on laryngology state that the condition is very rare.

Common Forms and Sites.—The most frequent forms of stenosis are webs or bands stretching across the glottic chink and uniting the cords. The anterior commissure is the common site. A web may extend as far as the middle of the glottis and is usually crescentic on its free edge. Although the color of the web closely resembles the vocal cords and may appear translucent, McKenzie found that the apparent "web" is usually but the flat upper surface of a somewhat thick, wedge shaped mass of tissue. Although webs usually unite the vocal cords, Kyle states that webs at times bind together the ventricular bands.

Embryologic Development of Vocal Cords and Occurrence of Malformations.—Through the courtesy of Dr. H. E. Radasch,‡ professor of histology and embryology in the department of anatomy, Jefferson Medical College, the following data is submitted relative to the embryologic development of the vocal cords and the occurrence of certain anomalies. "In embryos of the fifth week (5 mm. to 6 mm. length) the laryngeal groove is bounded on each side by a rounded eminence called the arytenoid swelling. Ventrally (at the base of the developing tongue) these join to form a transverse ridge over the third and fourth branchial arches; this is the epiglottic ridge or fold. The arytenoid swellings and epiglottic fold form a ∩-shaped ridge that His called the

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†From the Jefferson Medical College Hospital, Philadelphia, Pa.

‡Personal communication.

furcula. The slit-like opening may be termed the "primitive glottis."

During the seventh week (about 17 mm.) of development each arytenoid swelling becomes kinked or bent near its middle so that the primitive glottis becomes T-shaped with the transverse portion at the epiglottis. At this time the arytenoid swellings fuse and the primitive glottis is completely obliterated. This is, apparently, a provision on the part of nature to prevent the passage of amniotic liquid down the respiratory tube during the early stages of its development. About the tenth week, or shortly thereafter, this glottic fusion becomes dissolved and the arytenoid swellings lose contact with the epiglottis ventrally; the epiglottis becomes oval in form.

At about the eighth week (during the period of fusion) the ventricle of the larynx is formed. This appears as a small lateral (dorsoventral) outpouching of the epithelium on the medial surface of each arytenoid fold or swelling. As the central epithelium of each outpouching disappears, a dorsoventral, slit-like cleft is developed, the future ventricle of the larynx. During the tenth to the eleventh week, as the arytenoid fusion is dissolved, this ventricular slit is bounded by a superior and an inferior fold; the former represents the plica ventricularis, or ventricular band and it is more widely separated from its fellow than the lower pair. The latter represent the plicæ vocales, or vocal cords proper, and the space between them is the definitive glottis. During the later stages of development the glottis gradually assumes the adult form—that is, the ventral two-thirds is the intermembranous or phonatory portion, while the remaining one third is the intercartilaginous or respiratory part.

Malformations or anomalies of the glottis are chiefly of the web, band or atresic type. The latter is very rare and is, of course, incompatible with life. Webs and bands are the common form of deformity. These may connect the vocal cords at their anterior or posterior portions, though the anterior are the commoner. At times the bands are at a higher level and connect the plicæ ventriculares or ventricular bands together. Webs may also be subglottic in position.

CAUSES OR THEORIES.

1. Trophoneurotic.
2. Inflammatory.
3. Heredity.
4. Embryologic theory of adhesions.

1. Trophoneurotic. In the earlier days, and even now, when a cause is apparently unexplainable, it is often attributed to a trophic or neurotic condition. This is practically on a par with "cryptogenic" or "idiopathic" origin. It means little in most instances.

2. Inflammatory.—Hansemann believes these webs and bands to be due to inflammatory conditions, possibly congenital lues. There is little to support this theory.

3. Heredity.—Much is blamed upon heredity, and these conditions are too few to draw any definite conclusions concerning its influence.

4. Embryologic Fusion Theory.—This is the soundest and really only tenable theory. It is definitely known that in the early stages of development the primitive vocal cords are adherent to each other. After a certain length of time this fusion normally dissolves and establishes the adult condition of ventricular bands, ventricles and vocal cords.

The generally accepted theory is that these webs and bands are due to a partial separation of the original fusion of the primitive vocal cords. The webs and bands are more common at the level of the vocal cords, as these folds remain practically in contact throughout fetal life; hence webs are more common in the anterior portion. Subglottic webs or adhesions and supraglottic bands are less common, as these parts are naturally more widely separated as development advances so that their fusion would dissolve much earlier than that of the glottic region. Yet the fusion in those regions may dissolve only partially, leaving webs or bands as a result.

Fein is not in favor of the fusion theory as a whole, since these bands, as webs, contain areolar, muscle and cartilaginous tissues. He believes that the epithelial fusion is minor and secondary, while the exuberant overgrowth of the mesodermal tissue is the

prime factor. His theory is rather weak, as one must bear in mind that the epithelial fusion must be first, since the mesoderm is nowhere exposed, and even if it was, it would have to bridge the gap between the separated primitive vocal bands or arytenoid swellings.

The probable explanation of these bands, webs and atresia is as follows: Epithelial fusion takes place in all instances. After a certain period (usually two weeks) this epithelial fusion dissolves and re-establishes the opening into the larynx with the attendant formation of ventricular bands, ventricles and vocal bands. If the epithelial fusion be thin and the growth of mesoderm be exuberant in this region, then the mesoderm could and would work its way through the thin epithelial layer to the opposite side, forming a bridge of mesoderm. If this bridge was complete from front to back, then atresia of the larynx would follow, as a mesodermal fusion never breaks down, as far as we know. If, however, this mesodermal overgrowth was only over one-half or two-thirds of the glottic area then a web would be the result; if the mesoderm broke through only in spots, then bands would result."

Symptoms.—The symptoms depend upon the degree of stenosis. An incomplete separation of the vocal cords anteriorly may cause no disturbance in phonation or respiration and may exist for years without discovery until the larynx is examined for some intercurrent affection. Large webs may seriously interfere with respiration and phonation. In other cases a laryngeal stridor may be the only symptom. This usually will be heard both on inspiration and expiration, whereas in so-called congenital laryngeal stridor this is observed only on inspiration.

The diagnosis can be very easily made by inspection of the larynx. This is readily accomplished, even in the newborn, with the aid of a direct laryngoscope.

Treatment.—This depends on the degree of interference with laryngeal function. If there is no dyspnea little need be done. Bands are readily removed and there is little tendency to recurrence. Webs, however, tend to recur by adhesion of the edges and may prove troublesome. Jackson advocates incision along one cord only. The cut edge of the web will usually rest below

the edge of the cord and healing will occur without adhesions. As soon as this is healed the web may be cut from the other cord and removed. Intubation tubes may be inserted after the web is excised. Repeated bouginage may be necessary.

Three cases of stenosis of the larynx are reported. Two of these were members of the same family. The condition was discovered by direct laryngoscopy only after other diagnostic possibilities had been considered. In the third case, the condition was not recognized until late in adult life, although laryngeal symptoms had been present for a number of years.

Case 1.—Female, aged 7 weeks, had been dyspneic since birth, and stridor was present both on inspiration and expiration. The dyspnea was increased when crying. The voice was normal. There was no difficulty with swallowing. No gross developmental abnormalities were observed. A roentgen study of the chest had been made shortly after birth; this indicated the presence of an enlarged thymus gland. Appropriate treatment was carried out without apparent benefit. The child was then sent to the bronchoscopic clinic for examination. A direct laryngoscopy was performed without anesthesia. The mucosa of the entire larynx was found moderately inflamed. The anterior two-thirds of the glottic chink was found obliterated by a web which united both vocal cords and was crescentic along its posterior border (Fig. 1).

Tracheotomy was performed under local anesthesia and a No. 1 full curved Jackson tracheotomic cannula was inserted. As soon as the wound healed an attempt was made to dilate the larynx by the direct method, using triangular bougies. The web offered marked resistance, so it was excised, using a triangular punch forcep. Frequent direct laryngoscopic dilatations were necessary to maintain the lumen. The edges of the cords ultimately epithelialized and the patient was decannulated.

The patient is now 3½ years of age and has a normal voice. The larynx appears practically normal, although the edges of the vocal cords are thick. There is no dyspnea.

Comment.—McKenzie pointed out that simple excision of a web affords only temporary relief, as the web promptly reforms. Repeated dilatations were necessary in this case to maintain the

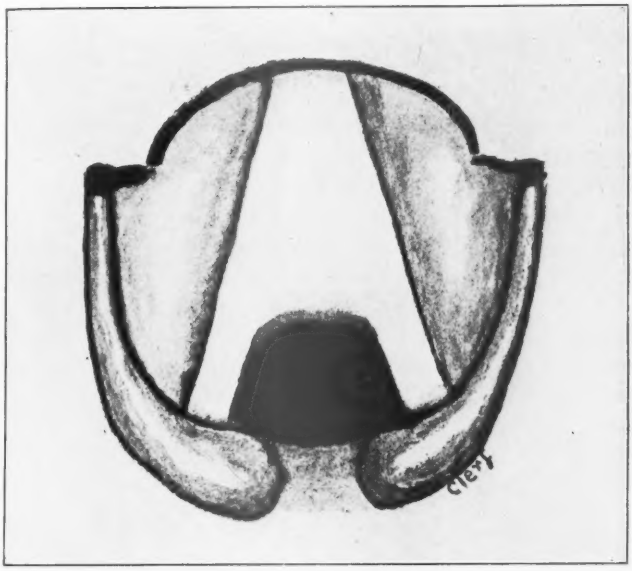

Fig. 1. Sketch of larynx showing web stenosis involving anterior two-thirds of glottic chink in the case of a child, aged 7 weeks. Posterior border was crescentic and was quite thick. It was necessary to remove the web. Simple bouginage was ineffective.

lumen secured by excision of the web. The case would very probably have recovered more promptly if the plan of treatment of webs advocated by Jackson had been employed.

Case 2.—Male, aged 2 days, a younger brother of the patient referred to in Case 1, was sent to the bronchoscopic clinic because of marked dyspnea, aphonia, difficulty with nursing and frequent attacks of cyanosis. The cyanosis was temporarily relieved by removal of secretion from the mouth and throat. A roentgen diagnosis of some enlargement of the thymus gland was made and appropriate treatment had been given on the day following the patient's birth.

The dyspnea, aphonia and attacks of cyanosis suggested some disturbance in the airway and direct laryngoscopy was promptly done. There was found complete obliteration of the membranous

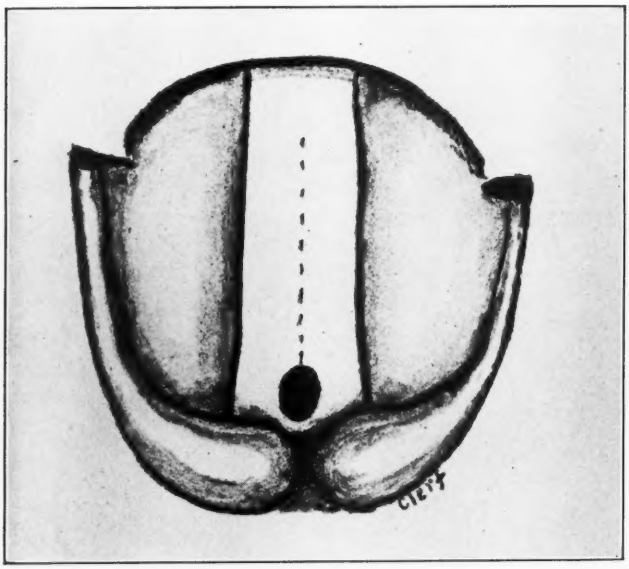

Fig. 2. Sketch of larynx showing union of membranous portion of vocal cords in an infant, aged 2 days. The airway was 1½ mm. in diameter. The cords were separated along the midline shown in drawing as a dotted line.

portion of the glottic chink by a union of the vocal cords in the midline. The cords appeared white and seemed to be wider and apparently longer than normal. The airway, a small rounded opening, about 1½ mm. in diameter, was found at the posterior commissure (Fig. 2). The arytenoids were slightly separated and their range of motility was greatly limited. With the removal of secretion from the pharynx the patient seemed to secure sufficient air. It was unable to cry. With a laryngeal knife, the point of which was inserted into the minute laryngeal lumen, an incision was made in the direction of the anterior commissure along the midline for a distance of about 8 mm. The incised membrane appeared to be thin and there was practically no bleeding. Following removal of the laryngoscope the child cried lustily. The dyspnea was completely relieved and there was no

cyanosis nor was there difficulty with taking of food. Three days later there was slight dyspnea. At direct laryngoscopy the vocal cords were found adherent anteriorly and the airway was greatly diminished. The cords were separated by bouginage. On the following day dyspnea again recurred and tracheotomy was done. The infant was then one week of age. Repeated laryngoscopic dilatations were necessary to maintain the laryngeal airway. The patient was ultimately decannulated, the tracheotomy fistula was closed by plastic operation and he was discharged at the age of 15 months. The larynx appeared practically normal except for some thickening of the vocal cords; the voice was normal.

Comment.—The membranous vocal cords appeared to be glued together; the airway corresponded to the cartilaginous portion of the cords. The difficulty in this case was to keep the cords separated. Although tracheotomy permitted the cords to remain approximated it was imperative because of dyspnea. Introduction of an intubation tube, as recommended by many authors, would have been effective in keeping the incised edges of the cords apart. I believed that repeated laryngoscopic dilatations would ultimately serve the same purpose and these could be safely carried out.

Serologic studies of the blood in Cases 1 and 2 were negative for lues. The parents also were examined with negative findings.

Case 3.—Female, aged 39 years, was referred because of hoarseness and dyspnea, particularly on exertion. Her general health had always been fairly good, although there was a history of frequent colds. She has always been underweight and there was dyspnea on marked exertion. There was no history of diphtheria, no operations on the larynx and no injuries. There was no evidence of pulmonary tuberculosis. The present symptoms began six years ago with slight huskiness following an acute cold. The symptoms gradually became more marked and now the patient was hoarse, there was dyspnea on exertion, loss of weight and inability to sleep well. A general physical examination failed to reveal any evidences of systemic disease. The laryngeal picture was very interesting. The epiglottis was of the overhanging type and interfered with a good view of the larynx—in fact, one could see only the arytenoids. This accounted for the failure

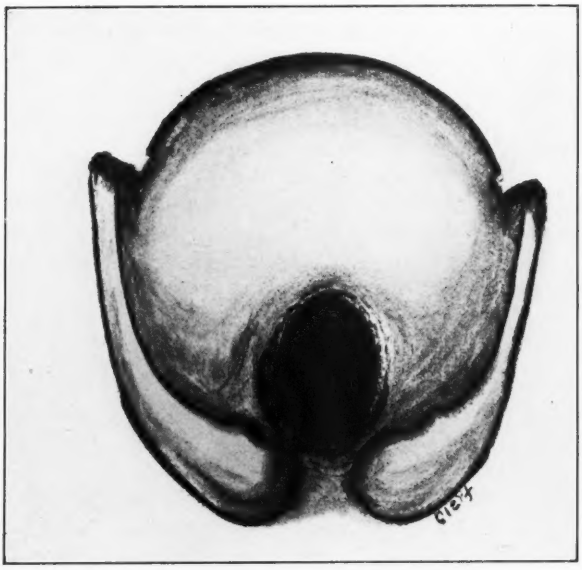

Fig. 3. Sketch shows unusual web-like stenosis of supraglottic larynx occurring in a woman, aged 39 years. The ventricular bands were fused together in their anterior two-thirds and the vocal cords were not visible until after the web had been incised.

of many laryngologists who examined the patient to correctly diagnose the condition. At direct laryngoscopy the vocal cords were not visible. The supraglottic larynx was almost completely closed by a diaphragm of tissue continuous with the ventricular bands; posteriorly there was a rounded opening which constituted the airway. On phonation, with the direct laryngoscope in position, the right vocal cord could be seen. The movements of the arytenoids were normal. There was nothing observed to suggest that the web was of cicatricial origin (Fig. 3).

With a laryngeal knife the web was incised to the left of the midline for a distance of 1 cm., and triangular bougies were passed. The glottic chink and vocal cords were normal in appearance. Dilatations were repeated five times at intervals of several weeks.

There was very prompt improvement, the voice is normal: there is no dyspnea; there has been a gain in weight. The laryngeal lumen is now roughly triangular in shape, and the vocal cords can be seen by mirror laryngoscopy.

Comment.—Webs uniting the ventricular bands are very rare and the question must always be considered whether they may not be of inflammatory or cicatricial origin. The appearances and the behavior of the web after incision, together with an absence of any possible acquired etiologic factor, seem to indicate that this was a case of congenital stenosis.

Recognition of these cases, particularly in infancy, is important. With the common practice of attributing respiratory difficulties in infancy to thymic gland enlargement there is little opportunity to determine the condition of the larynx, particularly in the fatal cases. Cases 1 and 2 were considered and treated as thymic enlargement, and it is safe to assume that Case 2 would not long have survived had the larynx not been inspected. Every patient with symptoms referable to the respiratory tract, particularly if these are dyspnea or aphonia, should have a direct laryngoscopic examination. Every adult with symptoms suggesting some disturbances of laryngeal function should be examined by indirect laryngoscopy. If this cannot be performed, then direct laryngoscopy is indicated.

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LXIV.

SUDDEN BILATERAL PARALYSIS OF THE LARYNX IN TUBERCULOSIS, WITH REPORT OF TWO CASES.*

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Unilateral recurrent paralysis of the vocal cords occurring in the tuberculous larynx is not particularly uncommon. St. Clair Thomson has observed eleven cases of unilateral paralysis due to pressure of a tuberculous lesion on the recurrent nerve outside of the larynx, five of which completely recovered, whereas two other cases persisted for three or five years.[1] Tensor paralysis or tensor paresis is quite frequent in tuberculosis and is said, by some authorities, to be an early manifestation of laryngeal involvement.

Bilateral recurrent paralysis in a larynx invaded by tuberculosis must be quite uncommon, as I have been unable to find record of any other cases. Paralysis of the vocal cords is usually "discovered" either because of some disturbance of phonation or in the course of a routine examination of the throat. Both of the cases herewith reported I was able to observe before and very shortly after the vocal cords lost their function.

Harry F. Davies, in an analysis of forty-nine cases of paralysis of vocal cords found seven cases occurring in patients who had pulmonary tuberculosis but no laryngeal lesions. These were all unilateral cases.

In his series were five cases of bilateral recurrent paralysis. One case was due to cerebral neoplasm, two cases due to carcinoma of the thyroid gland and two of undetermined origin.[2]

In 150 cases of recurrent paralysis reported by Avellis, twelve only were bilateral.[3]

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*Presented before the American Laryngological Association at Atlantic City, June 17, 1931.

A complete bilateral paralysis of the vocal cords suggests a lesion situated either centrally (in the medulla oblongata), one lesion massive enough to involve both vagus nerves above the ganglia of the trunk, of two lesions, one on each nerve above the ganglion, in which cases there would be anesthesia of the larynx in addition to immobility of the vocal cords. In the two cases reported here, there was no anesthesia of the larynx, consequently lesions above the trunk ganglia of the vagus nerves must be ruled out. An unusual occurrence was that both sides of the

Fig. 1. Case 1.

A. Larynx showing the tuberculous spindle cord.
B. The same larynx at the time of tracheotomy showing the vocal cords in adduction and fixed.
C. The same larynx after the vocal cords have assumed the cadaveric position. The tuberculous lesions have healed.

larynx were paralyzed simultaneously and it is difficult to conceive of two lesions causing pressure on the nerves at the same time.

REPORT OF TWO CASES.

Case 1.—A woman, aged 31 years, whose general health had been good until 1921, when she began with symptoms of pulmonary tuberculosis. Under sanitarium treatment, the tuberculosis was arrested. In 1924, she became pregnant, with the result that the pulmonary disease lit up. Her voice became husky a

October, 1926. The aggravated pulmonary condition had improved by the time the laryngeal symptoms were noted.

In November, 1926, the lungs were in fair condition. The nose and throat were negative except for the larynx. The right vocal cord was inflamed and spindle-shaped; the movements of both cords apparently were normal, but, due to the shape of the right cord, the voice was hoarse. The remainder of the larynx was negative. Sputum contained tubercle bacilli. The Wassermann was negative.

By March 9, 1927, the right vocal cord had greatly improved under vocal rest and chaulmoogra oil instillations. No surgical measures were employed on the larynx nor its environs.

On April 20, 1927, the right vocal cord again began to assume the spindle appearance, but movements of both cords were normal, although hoarseness persisted. During the following week the patient rapidly became dyspneic. The dyspnea was very marked on April 29, 1927, for a few hours, and then gradually subsided within two days. The patient was not seen by a laryngologist at this time, as she was away from Philadelphia.

On May 3, 1927, she was taken to the Jefferson Hospital because of a return of the dyspnea, which at this time was quite marked. At this time a diagnosis of bilateral recurrent paralysis of the vocal cords was made.

Laryngeal examination, May 3, 1927, by Dr. Chevalier Jackson, in consultation, showed a tuberculous infiltration of the right vocal cord; both vocal cords lie in adduction, leaving only a small slit between. The arytenoids tilt forward and inward and are motionless on phonation and respiration. The voice is aphonic. Externally the neck in the region of the larynx seems slightly fuller than it normally should. Laryngologic diagnosis: Bilateral paralysis of the larynx. Tuberculosis of the right vocal cord.

Neurologic examination by Dr. Edward Strickler was as follows: An examination of the nervous system is entirely negative with the single exception of the toes' plantar response and the throat condition. I feel sure it cannot be referred to a neurologic lesion.

X-ray report by Dr. Willis F. Manges: The larynx is irregularly calcified and it is very difficult to determine whether there is

anything such as a fracture present. I don't believe that I should be justified in making a diagnosis of fracture of the cartilages of the larynx, but there is a line appearing above the cords which might possibly be a line of fracture through the partly calcified cartilage.

A later X-ray showed the following: There is considerable cloudiness of the shadows of the larynx as if there were enlargement of the soft parts. There is a fairly widespread but not extensive tuberculosis involving both upper lobes and to a slight extent the lower lobes. There is a slight adhesion to the left diaphragm. The tuberculous process is perhaps not well healed.

On May 5, 1927, it became necessary to do a tracheotomy, as the dyspnea became urgent.

By September, 1927, the tuberculous right vocal cord had healed completely and the vocal cords had assumed the cadaveric position. The patient was able to breathe quietly when she placed her finger over the tracheotomy tube, but was unable to wear a cork in the tube for any length of time.

On December 28, 1927, she developed a small ulcer on the left side of the posterior surface of the epiglottis, which persisted for about four months, since which time the larynx has remained normal except for the paralysis of the arytenoids and vocal cords, which remain in the cadaveric position. The patient is still living and wearing a No. 5 tracheotomy tube.

Case 2.—A woman, age 23, with a history of tuberculosis involving the left hip joint in early childhood, with a flare-up in later years. During the past three or four years, she has had cough, expectoration and febrile disturbances suggesting a progressive pulmonary lesion. The sputum has been positive for tubercle bacilli.

About January 15, 1930, the patient complained of soreness in the throat. She had been hoarse for about a month previous to the soreness. There was no dysphagia.

General physical examination January 25, 1930, by Dr. Elmer H. Funk:

Far advanced, bilateral, active pulmonary tuberculosis with signs of extensive infiltration throughout the left lung and consolidation of the upper lobe with marked cavity formation. Ex-

tensive scattered infiltration of the right lung with multiple small cavities at the right apex. There are pleural bilateral adhesions, those on the left side being most marked.

The following is the report of the X-ray examination, made by Dr. Louis R. Wiley on November 25, 1929:

Stereoscopic films made of the chest, with patient in the prone position, show the heart to be displaced greatly to the left. On the lateral view we note adhesions between the left diaphragm and under the surface of the pericardium. This probably accounts

A B C

Fig. 2. Case 2.

A. Showing tuberculous ulceration and infiltration of the posterior wall, both vocal cords and the left ventricular band.

B. Same larynx at the time of the tracheotomy, showing partially healed tuberculous lesion at the posterior end of the vocal cords. The vocal cords lie near the middle line and are fixed.

C. Same larynx showing the cords in the cadaveric position, all the tuberculous lesions having healed in the meantime.

for the displacement of the heart over the left. The right diaphragm appears to be free.

The right lung shows the presence of a large cavity formation extending from the upper border of the second rib to the lower border of the third rib, about one inch from the periphery of the lung. This cavity appears to be about one and a half inches in diameter surrounded by a dense wall nearly a quarter of an inch thick. The remainder of the lung shows an increase of bronchial thickening throughout, which thickening extends well out to the

periphery. Along the course of these various linear markings we note the presence of numerous calcified glands. The left lung shows what appears to be the presence of a large cavity in the upper lobe extending from the apex to the upper border of the third rib and from the left sternal line outward to within three-quarters of an inch of the periphery of the lung. This area also appears to be encapsulated but not nearly to such a marked degree as the cavity in the right lung. The remaining portion of the left lung appears to be dense, due to a thickened pleura over this entire area. We are unable to demonstrate the presence of any effusions.

Laryngeal examination revealed a tuberculous infiltration of the posterior wall of the larynx and tuberculous ulceration of both cords posteriorly. There was also a shallow tuberculous ulceration of the left ventricular band.

Rhinologic examination revealed a tuberculous ulceration on each side of the septum. The ulcerations of the septum healed up promptly under chaulmoogra oil frictions in about one month. The ulceration on the ventricular band cleared up by April, 1930, under chaulmoogra oil instillations, but the infiltration of the posterior wall of the larynx and the vocal cords in the region of the vocal process of the arytenoids persisted. The movements of the vocal cords were normal on breathing but did not approximate completely on efforts at phonation. There was no improvement in the pulmonary condition, and the patient continued with cough, expectoration and elevated temperature.

In the early morning of May 5, 1930, the patient was awakened from sleep struggling for breath. Dr. E. H. Funk was called and under his administration she recovered, although the marked dyspnea persisted. She was taken to the Jefferson Hospital, where a tracheotomy was performed.

Mirror examination of the larynx just previous to the tracheotomy revealed both vocal cords lying close together near the median line and motionless. A diagnosis of bilateral recurrent paralysis of the larynx was made. This was confirmed by Dr. L. H. Clerf.

The patient was discharged from the hospital May 29, 1930. No active treatment to the larynx was done following the tracheotomy. The larynx was observed twice a week and showed a gradual widening of the glottic chink until August, 1930, when the cords had assumed the cadaveric position and the patient was able to wear a cork in the tube for short periods of time. In the meantime the tuberculous lesions in the larynx cleared up.

The pulmonary lesion progressed and the patient died November 2, 1930. The laryngeal tuberculous lesion remained healed up to the time of death.

COMMENT.

In Case No. 2, abduction of the vocal cords during respiration was normal up to within two days preceding the urgent dyspnea. At the time of the tracheotomy, however, the vocal cords were perfectly motionless and remained near the median line. In this case complete adduction of the vocal cords was hampered by the tuberculous infiltration of the posterior wall. It was possible to observe the first stage of recurrent paralysis before the power of adduction failed in both cases. The widespread and destructive lesions in the upper lobes could cause pressure on the recurrent laryngeal nerve on either side, due to thickened pleura at the apices, and I have observed several cases of unilateral paralysis of the larynx in tuberculous patients apparently due to this cause, but it is rather unique that both cords should become paralyzed by apical adhesions simultaneously. There is a possibility, of course, that the left recurrent nerve might have been injured somewhere along its course in the mediastinum.

In Case No. 1, there was X-ray evidence of enlargement of soft parts in the region of the larynx and external evidence of swelling of the neck which could account for injury to the recurrent laryngeal nerves.

In the second case the neck was very thin and the larynx easily palpated, but there was no evidence of enlarged cervical glands or other swelling. Neither case could be caused by a central lesion, because sensation was not abolished and the lesion must have been on the recurrent laryngeal nerve itself.

In both of these cases healing of the tuberculous lesions occurred after tracheotomy. This must have been due to the complete vocal rest obtained by the paralysis of the larynx and tracheotomy combined.

1923 SPRUCE STREET.

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LXV.

FACTORS INFLUENCING THE COURSE OF MASTOIDITIS IN INFANCY.*

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This paper represents a study of the mortality rate in this clinic, of infants whose cause of death has been diagnosed as acute otitis media or mastoiditis, with the idea of determining, so far as possible, the factors entering into their deaths. Recommendations based on this study for lowering the mortality rate are offered herein. These observations include all instances of the disease in patients under two years of age at the infants' ward of the Children's Hospital during the years of 1926 to 1930 inclusive. Most of the infants under observation were treated for various other conditions, such as syphilis, birth trauma, pyelitis, etc., as well as for mastoiditis. Those with uncomplicated surgical mastoiditis are admitted to the Otolaryngology Department. A need for the present study was suggested by the records of infants with mastoiditis during the years of 1924 and 1925, during which period were found a total of 16, 12 of whom were operated upon, with a mortality rate of 67 per cent.

During the past four years, 200 infants with mastoiditis have been observed. Eighty-nine, or 44½ per cent, were operated upon and 41 of these died. There were 31 deaths in the nonoperated group, this group including in most instances those having a much milder form of the disease. Seventeen of the patients operated upon had terminal conditions such as: marked hydrocephalus, encephalitis, primary bronchopneumonia, septicemia or sinus thrombosis before operation. These patients properly should not be considered in compiling mortality statistics dependent upon

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mastoiditis. A careful attempt was made to be unbiased in borderline cases, and when there was a question of doubt as to a case it was not considered in this group. In eliminating the above, we found a mortality rate of 34 per cent for the four years.

Numerous recent papers have called attention to the association of diarrhea of parenteral origin with mastoiditis, so the patients were divided into two main groups, namely, mastoiditis without diarrhea, and mastoiditis accompanied by diarrhea, emeses and other unusual manifestations. It was quite evident that this latter condition was quite different in course and indicated treatment from the usual mastoid infection. The death rate in this second group was found to be much higher than among the babies without diarrhea, or 39 per cent. Due to the higher death rate, this study concerns itself particularly with group two.

The diarrhea associated with mastoiditis varied all the way from a slight increase in the number and a tendency toward liquid stools to a marked diarrhea of eight to twenty mucous stools per day, with excoriated buttocks. This extreme condition, as described by Jeans,[1] is the syndrome commonly known as cholera infantum and presents the following picture: The onset is sudden, there is rapid loss of weight, fever, dehydration and appearance of intoxication. The intoxication is characterized by drowsiness or stupor and pallor with a grayish hue. The blood leucocyte count is from 13,000 to 30,000 or more. Vomiting and food refusals occur frequently and at times to a marked degree.

Fortunately, this syndrome occurred in only a small proportion of the infants with diarrhea and mastoiditis. There were six in 1926, two in 1927 and five in 1928. It is interesting to note that there have been none since. Nine had mastoidectomies performed with a mortality rate of 44½ per cent. One died without operation. Milder forms of diarrhea associated with mastoid infection were noted in 92 of the 200 cases observed.

Of the complications causing death following mastoidectomy in infants with diarrhea, the following data was procured:

Bronchopneumonia	43 per cent
Postoperative collapse	27 per cent
Septicemia	23 per cent
Lateral sinus thrombosis	7 per cent

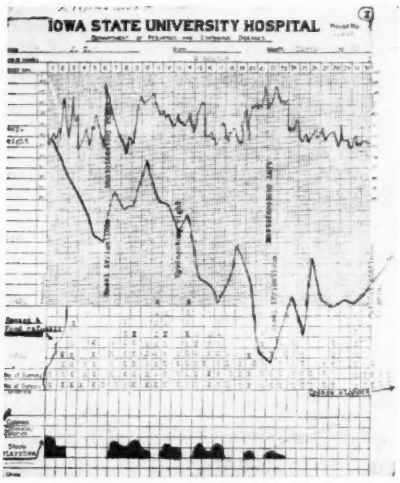

Chart 1.

Typical weight loss in cholera infantum with mastoiditis, due to emeses and food refusals rather than diarrhea.

In observation of the group with bronchopneumonia, several factors stand out clearly. The first and most important of these is the poor general condition of the patient at time of operation. Most of these infants came from indigent families and showed evidence of neglect. Many, apparently, had been undernourished since birth; and they were rarely without an accompanying ailment, such as lues, retarded mentality, birth injury or skin lesions. Furthermore, they were seldom seen early in the disease, but rather after the child was considered desperately ill. Lengthy operation seems to be the next most important contributory factor to this high percentage of bronchopneumonia. Obviously, infants of this type are poor risks, especially when marked diarrhea and dehydration are present. Although no arbitrary time limit can be set, the aim of the operator should be to perform as rapid and thorough a mastoidectomy as possible. Lastly, the incidence of bronchopneumonia following operation under local anesthesia

has been much less than when general anesthesia was the usual procedure.

The next most common immediate cause of death was postoperative collapse. These patients seemed unable to withstand the shock of operation and died within ten minutes to twenty-four hours following. The same factors which might aid in causing the high mortality in bronchopneumonia would naturally act here as well. However, it is apparent that early diagnosis and treatment are the chief factors in lowering the number of deaths due to postoperative collapse. This is particularly true with respect to patients with cholera infantum. Since this condition, which is primarily one of very rapid dehydration, is felt to be due to infection outside of the intestine, formula modifications and medical treatment to the bowel have little effect. Transfusions and fluids by vein tend to check this rapid weight loss in some instances, but in this series of cases no marked relief occurred until drainage of the infection was instituted. Of all the various factors contributing to the high mortality, late diagnosis and delayed treatment were unquestionably the most outstanding.

The otologist and pediatrician must be unusually alert and cooperative in order to make a complete diagnosis in early and subacute cases. The symptoms are chiefly of a general character and unless the possibility of mastoiditis is kept in mind and repeated examinations are made the ear infection will be overlooked. This is especially true where lues, pneumonia or some other concurrent infection is present to which many of the symptoms may be ascribed.

Attacks of syncope and sudden death associated with mastoiditis have been noted by Jeans,[2] who describes them as follows: Three infants, who were doing fairly well from the pediatric standpoint, except that there were moderate food refusals and some diarrhea and occasional temperature, were found dead in bed within a period of three months. Autopsy in each instance showed only marked mastoiditis, which had not been previously diagnosed. Since then, with more careful observation, three more infants have been observed during syncopal seizures from which they were resuscitated. In each of these cases definite ear signs were present, including bulging of the posterior canal wall.

Following mastoidectomies, no attacks of syncope or sudden deaths have been noted.

The usual procedure in diagnosis and treatment is as follows: An attempt is made to elicit the history of a head cold prior to or at the beginning of the illness. It is surprising how frequently this information is offered voluntarily by the parents. A history of upper respiratory infection in other members of the family is of importance, as will be later shown. Spontaneous rupture of the drumhead seldom occurs and no ear symptoms are noticed before admission. Many of the earlier cases show the red bulging drumhead while later it is often still bulging but gray and lusterless, and usually present on both sides. In practically all of the acute cases with mastoid involvement bulging of the posterior superior canal wall is constant. Marked edema or subperiosteal abscesses in the region of the mastoid are rarely found in this group. Rhinoscopic examination usually reveals some

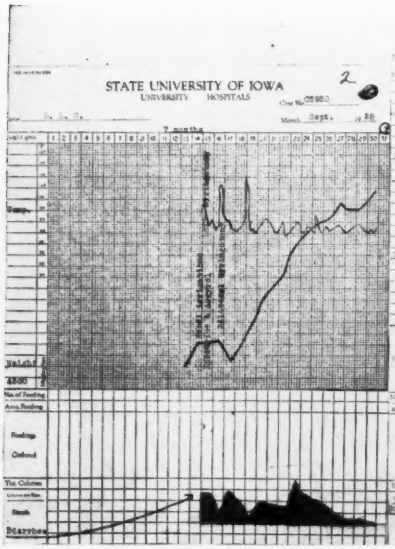

Chart 2.

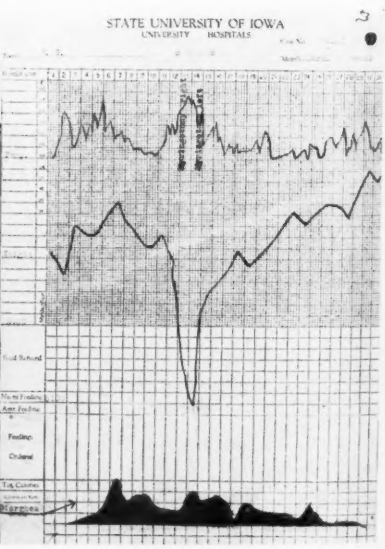

Chart 3.

Response of symptoms to myringotomies only.

evidence of infection in the nose or sinuses, and often there is a definite acute inflammatory reaction of the lateral and posterior pharyngeal walls.

When, and only when, there is definite pathology seen in the drumhead, myringotomy is done at once. Conservative treatment to the nose is also usually indicated. The patient is then observed for twenty-four hours, during which time fluids are given by mouth, and vein if necessary. In the meantime, routine X-rays of the mastoids and sinuses, reports of ear cultures, and other necessary laboratory data are procured. Unfortunately, in most instances this information is of much less importance than the physical findings. When there is decided improvement at the end of this period, treatment is limited to careful observation and supportive measures. In this series, over half of the cases observed at a relatively early time required no mastoid operation.

If the temperature remains elevated and there is continued weight loss, the need for immediate operation is indicated. Too much stress cannot be placed upon early diagnosis and intervention.

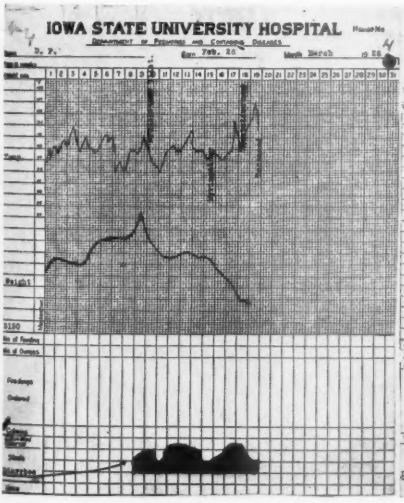

Chart 4.

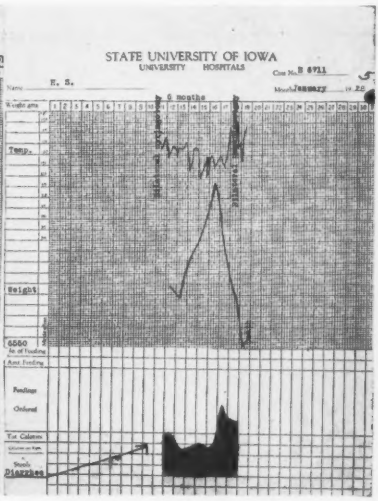

Chart 5.

No improvement following myringotomies. Mastoid operation delayed.

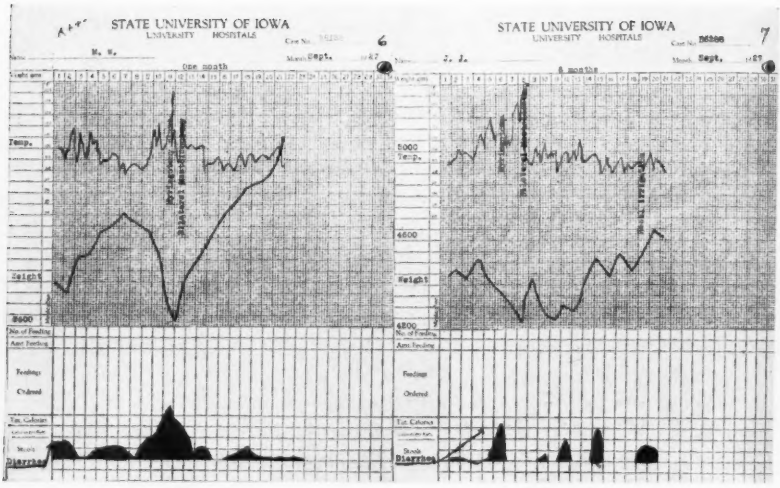

Chart 6. Chart 7.

No improvement following myringotomies. Early mastoid operation.

Following operation, the usual feedings and fluids by mouth may be started within an hour, along with other supportive pediatric measures.

Cultures taken at the time of myringotomy are of questionable significance. For example, cultures from the auditory canal before operation showed the following:

Staphylococcus albus	75 per cent
Streptococcus hemolyticus	15 per cent
Nonhemolytic streptococcus	6 per cent
Nonhemolytic staphylococcus	2 per cent
Unidentified organisms	2 per cent

Cultures from the exudate found in mastoid antra at time of operation on the same series of cases revealed:

Streptococcus hemolyticus	33 per cent
Nonhemolytic streptococcus	27½ per cent
Nonhemolytic staphylococcus	22¼ per cent
Hemolytic staphylococcus	10 per cent
Pneumococcus	5¼ per cent

Diphtheroids	1½ per cent
Gram negative bacillus	¼ per cent
Colon bacillus	¼ per cent

Streptococci were demonstrated in all cases complicated by sinus thrombosis and septicemia. Although in many instances of mastoiditis with marked diarrhea, streptococci were found to be present in the mastoid antra, just as typical diarrhea, dehydration and toxicity has been seen in several cases where staphylococci and other organisms were demonstrated. Prompt alleviation of all symptoms, observed repeatedly when proper drainage was obtained, has led to the conclusion that pus under pressure in a closed cavity, such as in the mastoid antrum and maxillary sinus, is the chief factor in causing this syndrome.

The communicability of mastoiditis in infancy has been frequently demonstrated. This year the pediatric wards have been relatively free from mastoid infection; of the 200 cases reviewed during the past four years, only 15 were seen in 1930. The following serves as evidence of the contagious character of this disease:

Within the past three months an infant was admitted to the general infants' ward of a near-by orphanage. He was not eating well, had diarrhea, excoriated buttocks and acute otitis media. Subsequently every one of the twenty-two infants in the home developed the same symptoms with an average of eight to ten stools a day, which in several instances were accompanied with much mucous. All recovered quickly following operation except one which developed intracranial complications. Pure cultures of hemolytic streptococci were obtained from the mastoid antra. Drs. Ott and Hands are preparing a more detailed description of this epidemic for publication. As a prophylactic measure, strict isolation is definitely indicated, particularly during the fall, winter and spring, when most of the diarrhea with mastoiditis has been found to occur.

From the standpoint of diagnosis and treatment, attention is called to the characteristic anatomic features of the infant middle ear and adnexa. Time does not allow for a detailed discussion, but the following facts are particularly interesting:

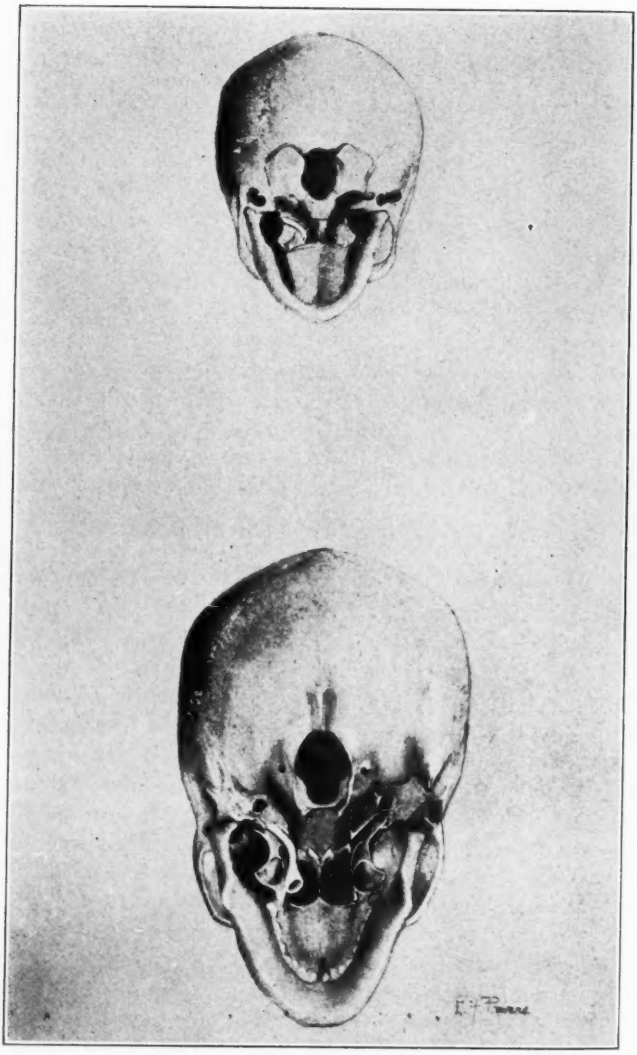

Figure 1.
Eustachian tubes in child and adult.

First, as *Dr. H. J. Prentiss points out, the eustachian tube is patulous, relatively much larger in caliber and more horizontal than in the adult. It is furthermore less than one-half the length of the adult canal.

As many of the infants of this group were artificially fed and the usual position is the dorsal one, it is easily understood how pus from the draining sinuses or regurgitated food would naturally enter the middle ear. Secondly, the ossicles in an infant are practically the same size as those of an adult, and with slight inflammation they may block the aditus and consequently the drainage of the antrum. Thirdly, the ethmoid cells of an infant

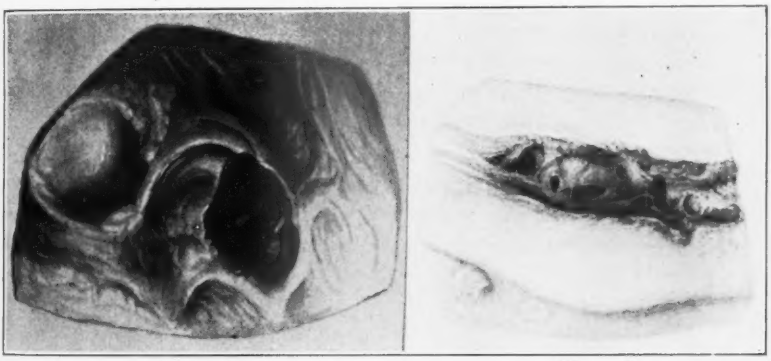

Figure 2. Figure 3.

Early embryonic tissue in infant tympanum before complete absorption has occurred, as drawn from specimens of Dr. G. Alexander.

are relatively large and well formed at birth, and the maxillary antra are usually present at a very early age. Fourthly, the infant tympanum, according to Alexander and others, is filled with mesodermal tissue of embryonic type. Following birth, resorption generally takes place but it may not be complete until as late as the fifth year. Thus many pockets are formed by fibrous strands of connective tissue and natural drainage is less likely to occur.

Too often the attention of the otolaryngologist has been directed solely to the mastoiditis. Infection of the paranasal sinuses

*Dr. H. J. Prentiss. Personal communication.

may have a very large part to play in causing and maintaining the mastoiditis, and in several instances sinusitis was the only infection associated with the characteristic symptom complex.

Patients illustrated by charts 8 and 9 are typical of a large group that have been under observation.

Chart 8 describes the course of a three-months-old infant who was admitted to the clinic because of weight loss, diarrhea and a head cold. Myringotomy had been done at home with no marked change in symptoms. With proper feedings and conservative treatment in the hospital, the diarrhea was reduced from ten to six stools per day. Later, due to an elevated temperature, weight loss and increased toxicity, mastoidectomy was done, and moderate infection was found. Five days later the diarrhea had increased to eighteen stools per day with a similar exaggeration of all other symptoms. The baby died within three days, and complete autopsy revealed only one positive finding, namely, the right maxillary sinus was filled with thick, ropy pus. A pure culture of pneumococcus was grown from the sinus aspirations.

The following month another infant of the same age and similar complaints came under observation (Chart 9). Here again it appeared that mastoidectomy, although moderate pathology was found at operation, had no apparent bearing on the accompanying symptoms, as over two weeks later there was a temperature of 107°, diarrhea of eleven stools per day and much weight loss. Due to the similarity in the clinical course of this infant with that of the previous fatal case, exploratory puncture of the maxillary antra was done. Thick pus was found and drainage instituted in the inferior meati.

Within three days, the temperature became normal, the diarrhea had practically ceased, and the child, whom we had recently considered moribund, was taking all of its feedings and the signs of toxicity had largely disappeared.

The following observations in regard to the relationship of mastoiditis and diarrhea have been noted:

1. When there is definite sinusitis accompanying the mastoid infection, as set forth above, improvement may not take place following simple mastoidectomy, until the sinuses, as well, have

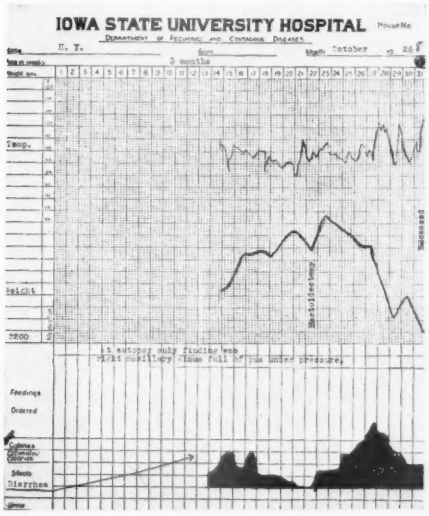

Chart 8.

Aggravated symptoms and death following mastoidectomy.

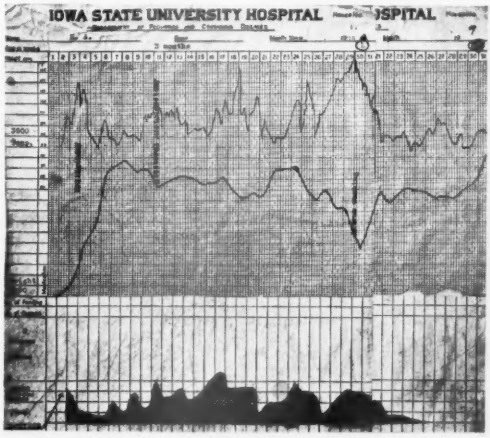

Chart 9.

Case similar to that shown by Chart 8. Infant recovered following drainage of maxillary antra.

been treated. It is possible that many of the so-called delayed recoveries and deaths took place because the sinusitis was not taken into consideration.

2. In the great majority of instances, diarrhea and weight loss ceased as soon as adequate drainage of the infection was established.

3. Necropsies have been done on 60 per cent of the fatal cases, and at no time has pathology of the gastrointestinal tract been found.

4. With the exception of an occasional epidemic, such as the one which occurred at the orphanage, mastoiditis with marked diarrhea occurs chiefly during the fall, winter and spring months, when upper respiratory infection is most prevalent.

CONCLUSIONS.

1. Mastoiditis in infancy is vastly different in symptoms, course and indicated treatment from that of the adult.

2. The mortality rate in infants having mastoiditis with diarrhea is higher than those having mastoiditis without diarrhea and requires particularly close observation and unusual treatment, i. e.,

   a. Early diagnosis and operative measures.
   b. Necessarily rapid operation with little trauma.
   c. A realization of the communicability of this disease, particularly in institutions.
   d. Recognition of the large rôle which paranasal sinusitis plays in these cases and the necessity for its active treatment.

3. The unusual symptoms associated with mastoiditis respond rapidly when all points of infection in the upper respiratory tract are found and adequate drainage is instituted.

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LXVI.

IVORY IMPLANTS FOR SADDLE NOSE: RESULTS IN FIFTY CASES.

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Ivory as an implant material in nasal corrective surgery has been used, along with other exogenous substances, off and on for years with varying success here and abroad. It connot be said to have received any marked degree of enthusiastic approval nor to any great extent have displaced autogenous cartilage and bone as a supporting material because of the prevailing prejudice against foreign bodies in general. While this attitude is to a certain extent logical, it must nevertheless be modified by certain mitigating reservations. Ivory cannot be classed with paraffin or celluloid because it is, after all, an animal epithelial appendage similar to dentine or dense bone and therefore more likely to be tolerated by the human host than the strictly chemical bodies just mentioned.

While it is true that an ivory implant must always remain inert within the tissues of the host and can in no way be assimilated or incorporated as an integral living mass, the same can be said, to a certain extent, of autogenous cartilage and bone. In the case of implanted cartilage we know that the cells die and the mass is either absorbed and replaced with fibrous tissue or else persists for a long time as an inert nonirritating foreign body. As for bone, the original structure is eventually absorbed and replaced by new bone tissue that grows out from the covering and adjoining periosteum. With ivory the most we can hope for is that the recipient tissues will tolerate its presence without reaction, in which case the implant remains indefinitely without change. It is, therefore, an advantage to know that the implant will perform its function as a supporting material without alteration in size or substance.

The other advantages claimed for ivory over bone and cartilage are as follows:

1. It is easily obtainable.

2. It is firm and light, smooth of texture, and easily shaped to required needs.

3. Its introduction is very simple and without risk.

4. It obviates the necessity of a second incision such as is necessary in obtaining cartilage and bone, with consequent scar and risk of infection.

5. In case of infection it is easily removed and can later be replaced with no more trouble than originally.

Having employed ivory since 1926, in fifty cases, we are prepared to draw some conclusions as to its value. Our records show that of the original fifty cases, forty-four have retained the ivory for periods of from one to five years. The results in our private cases were a little better than in our clinic cases, having lost two out of the twenty of the former as against four out of thirty of the latter. The complications encountered were as follows:

Primary Infection: Four Cases.—In two of these the implants were re-inserted after a suitable interval and have since been tolerated without reaction.

Spontaneous Extrusion.—One case, occurring three months after operation. This was a marked case of atrophic rhinitis in which the skin over the nasal bridge was very thin and closely applied to the underlying bone. It was not a favorable case to begin with.

Trauma Necessitating Removal of Implants.—Four cases, one after four years, one after three years, one after five years, and one in less than a year.

In all of these cases the implants had been borne without reaction during the intervening period, and the history in each instance was of a severe blow directly over the nose. The immediate result of the trauma in two cases was an infection necessitating the removal of the ivory. In the other two cases the implant was driven through the overlying skin in one and the nasal mucosa of the tip in the other.

The first complication that must be considered is that of primary infection. This is manifested, within 24 to 72 hours, by

Interlocking ivory implants after one year. The angle should be slightly more acute than shown.

redness and swelling of the overlying skin, slight temperature and headaches, and when observed must be followed by prompt removal of the implant. We have never seen any permanent damage result, as the subsequent drainage is followed by a return to the previously existing condition. Even the incision heals without much scarring and can be employed again at a later date for the re-insertion of the implant.

The cause of primary infection may be a low state of resistance in the host or faulty asepsis. We have never encountered primary infection where general anesthesia was employed and therefore are led to conclude that bacteria are introduced either through an imperfectly sterilized solution or from the skin surface by the injecting needle. In recent cases where we have employed local anesthesia we have been particularly careful in preparing the skin and have used novocain as dispensed in sterile ampoules. Also, we have tried to get along with a minimum amount of solution so as to distend the tissues as little as possible. The elevation of the dorsal tissues requires but little anesthesia, as they are much less sensitive than the skin of the columella. There is rarely any danger of hematoma within the tunneled space because these tissues are rather loose and readily opened by blunt dissection. Bleeding from the wound is usually controlled by the sutures. In the after care it is important that the wound be not sealed over. It is kept covered with yellow oxid of mercury, 1 per cent, which is applied several times daily. If crusts or scabs form along the line of incision they should be removed with peroxid and fresh ointment applied. The patient should remain quietly in bed for 48 hours and should be cautioned against touching the wound with anything but sterile gauze. The sutures are removed within two or three days. During the first 24 hours cold compresses will generally prevent much reaction and add to the comfort of the patient.

With regard to subsequent introduction of the implant in cases of primary infection we have found that an interval of at least 60 days should be permitted to elapse so that the tissues be fully restored to health and products of the injection be completely absorbed. Several cases of this kind that resulted in successful retention of the implant have convinced us that the original in-

fection and consequent removal was not due to any inherent fault of the ivory but to error of technic in its first introduction.

If the ivory has been successfully retained beyond the first week and all evidence of reaction has subsided, it is reasonable, in our experience, to expect that it will remain indefinitely. Where extrusion has resulted or removal of the implant necessitated at a later date we have found the cause in most cases to be a reaction following trauma to the nose. While ordinary movements of the face and nose or casual light accidental bumps are of no consequence and are borne without reaction or damage, a severe blow will either drive the implant through its protecting fibrous envelope to a point just below the skin, externally or the mucous membrane internally, where it will erode and have to be removed.

In one case, a young man who had borne an ivory implant for three years without reaction got into a fight, receiving a blow directly over the nose. The lower end of the implant appeared just beneath the skin of the nasal tip and within a week had eroded through. In another, a woman of 30 had borne a double implant for five years without any symptoms, when a fall resulted in swelling and redness which persisted until the ivory was removed.

At this juncture it is well to point out that a patient with atrophic rhinitis is not a good subject for ivory. We have found that the skin of the dorsum is too thin and the subcutaneous tissues too sparse to successfully retain a piece of ivory. Even though the danger of primary infection is not much greater than in a healthy nose, we have found that it takes only a minor form of trauma to start the implant on its way through the overlying tissues.

To insure a reasonable degree of success one must pay attention to the following factors:

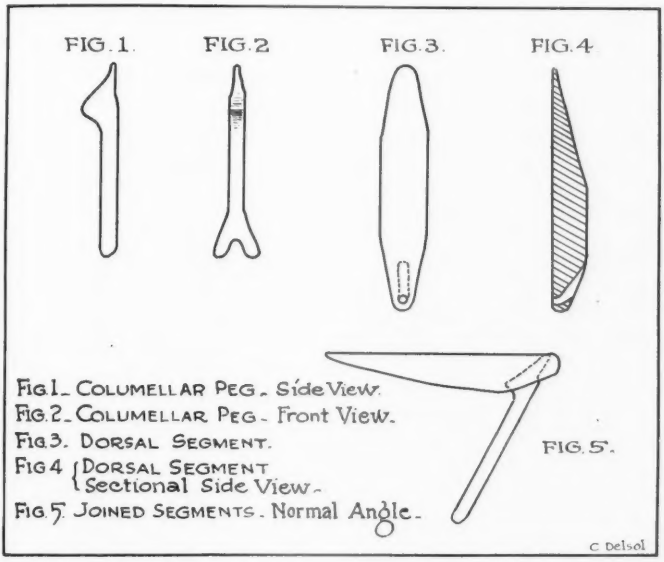
1. Accurate and careful preparation of the implant.
2. Thorough asepsis and a technic as simple and devoid of undue manipulation as possible.

The preparation of the implant is all important. It must be of such size and shape as to adequately fill the defect without subjecting the tissues to tension or pressure. There must be no stretching of the skin to a point where its circulation is inter-

fered with. Neither should the implant be sharp or rough. Its edges must be perfectly smooth all around, and there should be no lumps or ridges that might irritate. To accomplish this purpose, one must have a plaster model of the patient's nose as a guide in shaping the implant, and before its completion it should be tried on the outside to be sure of its size. By lifting the skin of the nasal dorsum and columella one can obtain a fairly accurate idea as to how much displacement it will tolerate without tension.

We have found the double interlocking implants of highest value in correcting a saddle nose and fallen tip caused by septal abscess. Here we have a condition the result of the loss of the greater part of the quadrangular cartilage. The dorsum below the bony bridge is sunken, the tip is lowered, and the nasal wings flattened. The entire cartilaginous part of the nose is soft and insufficiently supported by the weak columellar cartilage. Such cases can readily be restored to the normal contour by using two pieces of ivory, one under the skin of the dorsum and the other just behind the columella, the two interlocking at the tip.

The two pieces are cut from a block of ivory by means of a jig saw and shaped on a grindstone. The dorsal segment is tapered at its upper end and cupped slightly so as to ride easily over the bony nasal bridge and merge smoothly toward the root of the nose. The lower end of this segment must not be too broad, else it will spread the tip beyond its normal. A hole is drilled at this end to receive the peg of the columellar segment at an angle to conform to the normal angle between the columella and bridge of the nose, which varies in different individuals. The accompanying diagrams indicate the average inclination of this hole and also show a slot or groove which is excavated from the lower surface of the dorsal segment to receive a key-like projection in the columellar segment. The purpose of this arrangement is to distribute the support of the dorsal segment over a larger surface of the columellar segment and also prevent the latter from rotating. The hole and slot are slightly larger than the corresponding peg so as to permit a fair degree of play between the two segments without danger of their separation. Thus the normal displacements and movements about the lower end of the nose may be accomplished without discomfort and without rendering the



implants conspicuous. After the pieces have been accurately shaped and fitted they are smoothed off with sandpaper discs and polished on a buffer.

The technic of the operation is simple. Thorough preliminary cleansing of the skin of the nose, lip and face is accomplished in any of the approved standard methods. The face is draped, exposing only the nose and lip, and the tissues then injected. The incision in the midline of the columella from the tip to the philtum. The loose tissues of the membranous septum are opened by blunt dissection and then the tissues of the dorsum. We usually employ for this purpose a pair of long narrow scissors and open a tunnel beneath the skin of the dorsum as far as the root of the nose and only slightly wider than the implant itself. The implant should fit into this tunnel comfortably but without tension. If the tunnel is too wide there is danger of the implant slipping away from the midline. A tract is then opened behind the columella downward toward the nasal spine by blunt dissection. This also

should be only wide enough to receive the columellar segment which is to rest in front of the anterior nasal spine. Care should be exercised not to damage the periosteum covering the bone at this point. The implant must rest on uninjured periosteum and not on the bone direct.

The dorsal segment is now introduced and pushed far up into the prepared tunnel. Then the columella segment is pushed into place by first setting the lower end against the nasal spine. The lower end of the dorsal segment is then lifted by means of a flat dull septum elevator and the peg of the columellar segment slid along the surface of this elevator until it is opposite the hole which is to receive it. This makes it easy to lock the two segments. The incision is then closed by several interrupted horsehair or fine silk sutures.

The results in cases of successful retention of the implants have been most satisfactory. Patients state that they experience no discomfort and become utterly oblivious of the foreign body. We have seen two or three cases where the skin overlying the ivory became red, especially when exposed to the cold, but have been convinced in these cases that it was due to the skin being stretched too tightly over the implant, a fault that we carefully avoided in subsequent operations.

Instances where it became necessary to remove the implant, either on account of trauma or a desire to replace it with a better fitting segment, have demonstrated the tolerance of the tissues. On removing the pieces we have several times noticed a very definite fibrous envelope which had formed around the implant. This is doubtless a normal tissue reaction to the foreign body and is a distinct advantage insofar as insuring its retention. This fibrous capsule has never developed sufficiently in any way to alter the appearance or condition of the overlying skin in cases observed for a period of from three to seven years.

In conclusion, we desire to reassert our conviction that ivory is an admirable supporting material in cases of saddle nose and depressed nasal tip due to loss of cartilage, and that care in selection of the case, preparation of the material and strict asepsis in its introduction will yield a high percentage of success.

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LXVII.

UNILATERAL LOSS OF VESTIBULAR FUNCTION PROBABLY DUE TO EMBOLUS.

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Embolus to the inner ear, particularly with circumscribed, permanent, unilateral loss of function, is rare. I have found only one such case reported. Therefore, a case is presented.

The patient, a white female, of thirty-three years of age, was first seen in consultation September 22, 1930.

Chief Complaint—Dizziness.

History Present Illness—Began suddenly two days ago. Has been ill in bed since with nausea. More comfortable lying on the right side. No symptoms referable to ears. No other symptoms of note. Sudden turning of head to the left brings on an attack.

Past Medical History—The one thing of practical importance is a pulmonary infection two months previously. (Family physician states this was proved bacteriologically to be a spirochoetal infection and that it cleared up with arsenic and bismuth therapy.)

Examination—A well nourished adult female who lies on her right side. The eyes were negative except for a well defined second degree mixed nystagmus to the right. Nose and throat examination showed no gross pathology. The ear tests done at that time at the bedside are recapitulated below:

Cochlear:		Right	Left
Bone Conduction	(MacKenzie Fork 190 d.v.)	Normal	Short 15 sec.
Big C	(By air)	Normal	Normal
c4	(By air)	Normal	Short 8 sec.

Caloric—Reactions within the bounds of normal on the right. When the left ear was irrigated with the head erect there was no vertigo and no change in spontaneous nystagmus. Apparently a marked decrease in function exists on the left side.

The patient was so ill no further examination was done at this time.

General—The family physician reported his examination as negative and urine and blood pressure normal. The Wassermann was negative.

Tentative Diagnosis—Probably an embolus to left internal ear in view of previous pulmonary history.

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The prognosis was given as good because compensation is usually rapidly established in unilateral loss of vestibular function. Bromides only were advised.

Subsequent Course—Two months later the patient was reexamined. The hearing tests then done are appended.

Cochlear:		Right	Left
Bone Conduction	(MacKenzie Fork 190 d. v.)	Normal	Normal
Big C	(By air)	Normal	Normal
c4	(By air)	Normal	Normal
Audiogram		Normal	Normal

Caloric—Entirely normal reactions on the right. All reactions (vertigo, falling, past pointing and nystagmus) were completely absent on the left with head erect and back.

Revised Diagnosis—Embolus of terminal blood supply to left vestibular labyrinth.

In this case we have what is usually lacking viz., a frank source of embolus due to a previous pulmonary suppuration. Not only do we have a source, but because of its location a ready entrance into the left heart and arterial circulation. In the late stage of healing, particularly after a destructive Vincent's infection, it would be relatively easy to get a small embolus from the lung.

In addition, these things speak for probable embolus: (1) Sudden onset; (2) sharply circumscribed, unilateral character involving only vestibular function; (3) permamency of injury, as shown by subsequent complete loss of vestibular irritability; (4) normal cochlear function, pointing to an end organ lesion. A neuritis would have probably involved both branches of the eighth nerve.

The terminal supply of the internal auditory artery is apparently subject to some variation. Piersol[2] gives the description of Siebenmann as follows: An anterior vestibular supplying the upper vestibule, the macula of the utricle, the saccule, and the cristæ of the anterior and horizontal canals; a cochlear giving two branches to the lower turn and a third branch to the middle and apical turns; lastly, a vestibulocochlear from the cochlear, or independently from the internal auditory, with a cochlear branch to the lower turn, and vestibular branches to the lower vestibule, the macula of the saccule, and crista of the posterior canal.

If this description is correct, we can only assume an embolus to the anterior vestibular branch which supplies most of the vesti-

bule and its appendages with enough disturbance of function to give the symptoms presented.

For practical purposes, we must assume two main terminal supplies—i. e., a cochlear and a vestibular, with probably some variation. In this case the vestibular portion, or major portion thereof, was probably blocked.

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LXVIII.

THE DEVELOPMENT OF THE ORGAN OF HEARING.*

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After fertilization of the ovum, segmentation and gastrulation, the embryo becomes, during the second week, a simple disc, covered with ectoderm and containing a central cavity (primitive gut) lined with entoderm. An axial thickening of ectoderm now makes its appearance on the dorsal surface. This thickening, the primitive streak, shows at its head end the primitive pit (future point of division between head and neck) and the primitive knot, where all three germinal layers are later fused. From the primitive streak is developed the middle germinal layer, mesoderm. The primitive streak recedes caudalward, and headwards there appears another thickening of ectoderm which quickly sinks into a groove—the neural groove. The edges of this groove become prominent and soon come together to form the neural tube or primitive central nervous system. Caudalward, between the primitive streak and the neural groove, is the neurenteric canal. The neural tube remains open, cephalad and caudal, until after the fourth week. These openings are the neuropores. Quite early, in embryos of 2.5 mm., the cranial end of the neural tube has expanded into the three primary brain vesicles, prosencephalon, mesencephalon and rhombencephalon. Just caudal to the rhombencephalon is the myelencephalon. In studying the development of the ear we are concerned chiefly with the latter division of the brain, which in the adult is represented by the medulla oblongata.

‡Fig. 1. Rabbit embryo (human age, ten days). At the caudal end of this embryonic disc is the primitive streak. The long dark line above is the neural groove, showing four mesodermal seg-

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ments on either side. Between the neural groove and primitive streak is the neurenteric canal. Cephalad, the groove has expanded and divided into what is faintly discernible as brain vesicles.

Fig. 2. Chicken embryo (human age, twelve days), showing the neural groove with its prominent edges, the neural folds, approximating. Ventral to the neural groove is the notochord. On either side is a mesodermal segment, showing laterally the caelom (primitive body cavity). Extending from the neural folds to either side is ectoderm. Ventral to the mesodermal segments is entoderm. Dorsal to the caelom is the somatic mesoderm, ventral, the splanchnic mesoderm. On either side of the notochord, between segments and entoderm, are the positions of the descending aortæ. One notes that the neural groove ectoderm is quite thick.

Fig. 3. Chicken embryo (human age, sixteen days). Transverse section showing the closed neural tube with the notochord. Ventrally is the pharynx (head end of primitive gut) with the pharngeal membrane. One descending aorta is noted between pharynx and neural tube. Surrounding pharynx and neural tube are the stellate mesenchymal cells. Ventral to the embryo is entoderm and ectoderm of proamnion.

From now on we are interested in the level of the myelencephalon: that portion of the primitive brain which corresponds to the medulla.

Fig. 4. Rat embryo (human age, twenty days). Transverse section through the hind brain. Note that the neural tube has expanded to large dimensions and that the brain wall is very thick. Ventrally is the notochord and on either side is a mesodermal segment. On one side may be seen a distinct projection of myelencephalon tissue. This is the first evidence of the acoustic ganglion. It is growing toward a small thickening in the ectoderm. This thickening is the auditory placode, the first sign of an internal ear. The specimen is cut in such a way as to show the placode on one side only.

Fig. 5. Transverse section (specimen presented by Prof. Max Bielschowsky) of an adult human medulla, at the level of the upper end of the inferior olive. The medulla corresponds to the

myelencephalon of the embryo. We see the beginning of the ramus cochlearis, the anterior cochlearis nucleus (nucleus accessorius acustici), just anterior to the corpus restiforme and buried in the trunk of the nervus cochlearis. This nucleus takes up most of the fibers of the nervus cochlearis. Some of the fibers, however, combined with others springing from the anterior cochlearis nucleus, curve laterally around the corpus restiforme as the lateral acoustic root and find an interruption posterior to the corpus restiforme in the nucleus cochlearis dorsalis (tuberculum acusticum). The continuation of the system of the cochlearis is carried out by the striæ acusticæ, which run along the floor of the fourth ventricle, toward the median line, crossing at different levels. Just anterior to the striæ is the small cell triangular vestibular nucleus (nucleus vestibularis parvicellularis). To the lateral side of this nucleus is seen, in transverse section, the spinal acusticus root, which may be divided into three parts: most median, the descending fibers of the vestibular, laterally the connection between the nuclei of the medulla and the cerebellum (tractus nucleocerebellaris), which touches the restiform body, and between the two, the large cells of Deiter's nucleus (nucleus vestibularis magnocellularis).

Fig. 6. Transverse section through an adult human medulla (specimen presented by Prof. Max Bielschowsky) at a higher level than Fig. 5. We first note the ramus vestibularis. Its chief mass of fibers is directed toward the small cell triangular vestibular nucleus, while a second part curves toward the large cells of Deiter's nucleus. Some of the vestibular fibers, together with fibers from Deiter's nucleus, take the dorsal course of the restiforme fibers; some reaching the small cell nucleus angularis and others passing to the three cerebellar nuclei. A bit of the facial nucleus is seen, the fibers of which surround the nucleus of the abducens, after which they form the ascending arm of the facial. This is situated just dorsal to the fasciculus longitudinalis posterior.

Fig. 7. Transverse section (specimen presented by Prof. Max Bielschowsky) through the beginning of an adult human pons, showing the continuation of the acoustic fibers. In the tegmental region is seen a bundle of fibers which originate in the region

of the ventral cochlearis nucleus, anteriorly, winding around the spinal trigeminus root and then curving in a bow toward the median line, where they cross. This bundle, which in animals with a shorter pons, lies exposed on surface, is the corpus trapezoides. In its lateral third is buried the superior olive. From the corpus trapezoides radial bundles extend into the olive anteriorly, while dorsally the so-called pedicle fibers enter. These dorsal fibers are the continuations of the striæ acusticæ. In this section are also seen remains of the large and small cell vestibular nuclei.

To return to embryology: A few days after the auditory placode appears in the wall of the myelencephalon there is noted a sinking in of the thickened ectoderm, to form the foveola labyrinthica.

Fig. 8. Rat embryo (human age, twenty-four days). Transverse section through the myelencephalon, showing on the left a foveola labyrinthica. There is an ever narrowing communication with the outside. At this stage the passageway is called the appertura vesiculæ auditivæ. Ventrally is the pharynx and sections through the first branchial arch. On the right the foveola has closed. We note that the wall of the foveola is several times as thick as the ectoderm from which it is derived.

At twenty-eight days or shortly before, the foveolæ close to form the auditory vesicles. They are, according to some textbooks, lined with columnar epithelium, filled with albuminous fluid and contain each an otolith. The next figure shows us that sometimes this is not the case, as far as the columnar cells are concerned. In some species of fish this stage of aural development remains the permanent organ of hearing.

Fig. 9. Human embryo (twenty-eight days). Frontal section. To the left are seen the rather regularly arranged cells of the limiting ectoderm. In the center is the auditory vesicle, the wall of which is composed of very disorderly ectodermal cells of various shapes, very few being columnar. There is no sign of an otolith, even though, of course, no decalcification was used. Around the vesicle is massed condensed mesenchyme. The condensation of mesenchyme is the first step in the development of the periotic capsule.

Fig. 10. Rat embryo (human age, twenty-eight days). Frontal section through the myelencephalon. We note on either side of the hind brain an auditory vesicle. These vesicles are lined with a much higher epithelium than in the human specimen and in very orderly arrangement. The vesicle on the right contains a small something which may be an otolith. Note that the brain is sending out, on either side, a projection: the acoustic ganglion. Surrounding the vesicles are masses of stellate mesenchymal cells in a loose mesh of delicate fibers. Ventrally is the pharynx lined with thin entoderm.

At thirty-one days or about that time, the auditory vesicle has thrown out a dorsomesial projection called the recessus labyrinthi. This projection from the cavity of the vesicle continues to elongate until, in the adult, it ends on the posterior surface of the pyramid in a sac: ductus endolymphaticus and saccus endolymphaticus.

Fig. 11. Rat embryo (human age, thirty-one days). Frontal section through the myelencephalon. To right and left of the hind brain are the primitive labyrinths at the stage of recessus labyrinthi formation. Dorsomesially each auditory vesicle shows a somewhat curved outpouching. These evaginations elongate to form the endolymphatic ducts. There is a bare suggestion ventrally of an inward curve (beginning of the processus cochlearis). Note that the auditory vesicles are surrounded by condensed mesenchyme and that each vesicle is accompanied, ventromesially, by the acoustic ganglion.

Fig. 12. Rat embryo (human age, forty-five days). Transverse section. The condensed mesenchyme about the labyrinth has become more orderly and sharply delimited in its relation to the remaining cephalic mesenchyme. Suggestions of cartilage matrix formation may now be discerned under the microscope. The periotic capsule has arrived at the precartilage stage. The otic vesicle shows a constriction at its center, which marks the beginning of division into the utriculus dorsally and the sacculus ventrally. Further we note that the mesial wall of the vesicle now shows a thickening, which is the beginning of the maculæ acusticæ. The processus cochlearis, which is in Fig. 11 was just suggested, is here well

under way and shows a first half turn. In the concavity of the cochlear crescent is a dark mass, the spiral ganglion. This collection of ganglion cells will develop along with the winding cochlea, following it in its final two and a half turns. To the left of the utriculus is an oval mass, the vestibular ganglion. Below the vestibular ganglion and mesially is a part of the facial nerve. To the left of the vestibular ganglion is the myelencephalon showing acoustic and facial nerve tissue protruding from its lateral wall. Lateral to the processus cochlearis are two small oval spaces, sections of semicircular canals, which, after being pinched off from the vesicle through compression of its walls, have grown out into the mesenchyme. Above and lateral to the utriculus are again sections of semicircular canals. Please note that, surrounding the membranous labyrinth, there is continuous precartilage, with no evidence whatever of perilymphatic spaces.

Fig. 13. Rat embryo (human age, forty-five days). This is the same situation as in Fig. 12 with a slightly larger magnification. Just as the utriculus and sacculus have already developed a thickening of one wall to form the maculæ acusticæ, so we note in the section of semicircular canal (lower ring) a thickening which is the first step in formation of a crista ampullaris. Around the three sections of canals we see precartilage. Mesially is myelencephalon.

Fig. 14. Human embryo, age 60 days. Horizontal section. The cochlea has grown to one and one-third turns. The epithelium is so darkly stained as to appear traced with ink. Surrounding the cochlea is precartilage, the periotic capsule, at this age. The cochlea is still a simple canal, with a suggestion of thickening of one wall. Looking closely at the semicircular canal, on the left, one notes around it a lighter area than the remaining precartilage. This areola is the beginning of differentiation of precartilage into syncytial reticulum. Soon the syncytial reticulum will be resorbed, leaving a perilymphatic or periotic space about the membranous canal. Exactly the same process occurs about the utriculus, sacculus and cochlea. The perilymphatic space about the utriculus becomes the cysterna perilymphatica; that about the cochlea, the two scalæ (vestibuli and tympani).

Fig. 15. The same human embryo as Fig. 14, showing on the right a part of the myelencephalon with the vestibular ganglion (Scarpa's) and nerve. To the left is the labyrinth with its capsule of precartilage, undergoing dedifferentiation about the canals. The two larger oval spaces are ampullæ with definite epithelial thickenings representing cristæ formation. No sign of cupola or hair formation is present. The two smaller openings (semicircular canals) show halos of beginning dedifferentiation.

Fig. 16. Rat embryo (human age, 65 days). The section is horizontal, showing the hind brain in the center with the cochleæ on either side. To the extreme left is an early stapes with the stapedial artery and Reichert's cartilage attached. The apparent spaces about the upper section of cochlea (right and left) are artefacts as dedifferentiation of precartilage and absorption of syncytial reticulum has not yet occurred. A very important change, however, has taken place in the epithelium of the cochlea, namely, a thickening which is destined to become the organ of Corti, limbus spiralis, etc.

Leaving the aural labyrinth for the present, let us study the development of the eustachian tubes, tympana and ossicles. These structures depend, for their existence, upon the previous development of the branchial arches and pharyngeal pouches, during the first weeks of life.

Fig. 17. Section of a tadpole showing the simple manner in which the branchial arches and pharyngeal pouches are formed. In the center of the head is a large cavity, the primitive pharynx (head end of primitive gut). The pharynx is lined with entoderm and surrounded by mesenchyme. On the right, the entoderm has grown into the mesenchyme, dividing it into regular masses which are termed branchial arches. The specimen is unevenly cut, else both sides would show the arches. The entoderm lined passageways between the arches are the pharyngeal pouches, the first pair of which become the eustachian tubes and tympana. Externally the ectoderm sinks in between the arches, forming the branchial grooves, the first pair forming the external auditory canals. The branchial arches form posteriorly, grow forward and finally unite ventrally. The first pair divide to form the maxillary and man-

dibular arches, the second pair, the hyoid arch. Now in the interior of the mandibular arch is differentiated from mesenchyme a core of cartilage which extends backward to the middle ear region to form the malleus and incus: this is Meckel's cartilage. In the hyoid arch is a similar core, Reichert's cartilage, which, extending dorsally, divides into an external branch to form the styloid process and a median branch to form a ring around the stapedial artery. This ring of cartilage becomes the stapes.

Fig. 18. Rat embryo, horizontal section (human age, three months). In the center is the pharynx, showing on either side, a eustachian tube (first pharyngeal pouch), extending outward and expanding into the tympanum. Posterior to each tube is a cochlea. Posterior to the cochleæ are the vestibular portions of the labyrinths. One notes that the tympana are filled with mesenchyme except for the open expanded tubal ends. Between the two ears is the myelencephalon.

Fig. 19. Rat embryo (human age, two months). Frontal section, showing the primitive nasal fossæ with turbinal prominences, precartilaginous nasal capsule, thick septum with vomeronasal organs of Jacobson, primitive choanæ, lateral palatine processes of the maxillary arches, enamel organs, primitive oral cavity with tongue pointing upward, and lastly the reason for this photomicrograph, the mandibular division of the first arch, showing Meckel's cartilage. This cartilage core extends backward to the tympanum on either side to form the malleus and incus. In the second or hyoid arch (not seen in this section) is another cartilage core, Reichert's cartilage, which, extending posteriorly, divides into one part for the styloid process and into another for the stapes. The stapedial process first throws a cartilaginous ring around the stapedial artery, then proceeds to form the head, etc.

Fig. 20. Rat embryo (human age, four months). The tympanum is fairly clear of mesenchyme at this level. Meckel's cartilage is in a state of atrophy, having diminished markedly in size. Still attached to it, dorsally, is the head of the malleus and body of the incus (one mass of cartilage in the beginning). The malleus and incus now show a distinct line of separation but a true joint, such as we have in the adult, has not yet formed.

Median to the tympanum is the labyrinth, showing the utriculus with its macula and with its nerve emerging from Scarpa's ganglion. Just anterior to Scarpa's ganglion is the facial nerve. It will be noted that the capsule of the labyrinth shows considerable bone formation and complete resorption of syncytial reticulum about the utriculus.

Fig. 21. The same specimen as Fig. 20, at a slightly different level and under higher magnification. Here can be seen more plainly the atrophy and vacuolization of Meckel's cartilage; also the conversion of periotic cartilage into bone.

Fig. 22. Rat embryo (human age, 3 months), horizontal section, showing the cochlea, ventrally, the cysterna perilymphatica dorsolateral to the cochlea. The syncytial reticulum of the cysterna is disappearing. To the left of the labyrinth is the tympanum still filled with mesenchyme. In the tympanum are several structures which engage our attention. Ventrally is an elongated space, the first pharyngeal pouch or eustachian tube. Just dorsal to it is Reichert's cartilage, which has thrown a ring around the stapedial artery and is now undergoing atrophy separation from the future stapes. Dorsally, we see differentiating out of mesenchyme the stapedial muscle and tendon, while ventrally is forming the tensor tympani muscle. There is as yet no bone formation in the capsule of the labyrinth, the dark areas being deeply stained cartilage. To the right is the myelencephalon. It will be remembered that Reichert's cartilage, seen in this specimen, is the core of the second or hyoid arch. In the center of the stapedial ring is the stapedial artery, an embryonic structure which will disappear before birth.

Fig. 23. Rat embryo (human age, 3 months). We now return to the further development of the cochlea. In our last view of this organ, we noted the even thickening of one wall. In this section the thickened wall of the cochlear canal has differentiated into an outer moderate thickening and an inner greater thickening. Between the two is already forming the canal of Corti, to be traversed later by the filaments of the nervus cochlearis. The inner thickening is to become the limbus spiralis, sulcus internus, membrana tectoria and the inner hair cells of Corti; the outer

thickening, the outer hair cells of Corti. Already the membrana tectoria has been differentiated. The core of the cochlea, the modiolus, is beginning to take form from ingrowths of capsule cartilage. Accompanying the cochlear canal is the spiral ganglion. The peripheral fibers of the nervus cochlearis are reaching toward the limbus (the canal of Rosenthal has not yet formed). The capsule of the cochlea is pure cartilage, there being as yet no evidence of bone formation. In the basal turn we note the partial disappearance of syncytial reticulum to form the scala vestibuli and scala tympani. Above the cochlea (ventrally) is the first pharyngeal pouch (eustachian tube), leading medially to the pharynx and laterally to the tympanum, in which may be seen a part of the malleus and incus. In the internal auditory meatus is Scarpa's ganglion with the vestibular nerve emerging from the myelencephalon. Note that the cochlear duct is oval on section.

Fig. 24. Rat embryo (human, age 3½ months). At this age we see the capsule of the cochlea, still cartilaginous. Syncytial reticulum has almost entirely disappeared, leaving well formed scalæ. The limbus spiralis and sulcus internus are well differentiated. The organ of Corti is taking form. Rosenthal's canal has appeared and is filled with cochlear nerve fibers from the spiral ganglion. The modiolus is almost adult in appearance. Note how the capsule has grown in between the turns of the cochlea. The membrana tectoria is plainly seen. Syncytial recticulum has been converted, peripherally, into the ligamentum spiralis. The outer wall of the cochlear canal, showing dark, has become the stria vascularis. One wall has thinned down and become the vestibular membrane (membrana Reisnerri). The chief mass of cochlear fibers may be seen in the internal auditory meatus. Ventrolaterally is the eustachian tube and tympanum. Note that the cochlear duct, on section, is no longer oval but triangular.

Fig. 25. Rat embryo (human age, 3½ months). Section through an ampulla of a semicircular canal, showing a crista ampullaris. To the left is cartilaginous capsule with endochondrium differentiated. To the right of this is periotic space showing remains of syncytial reticulum and fibers of the nervus utriculo-ampullaris, passing to the neuro-epithelium of the crista.

Resting upon the neuro-epithelium is a half developed cupola and hair cells. The membranous ampulla encloses the endolymphatic space.

Fig. 26. Rat embryo (human age, 3¾ months). A frontal section through the posterior aspect of the tympanum and labyrinth. Partial resorption of the mesenchyme of the tympanum has occurred. The promontory shows beginning ossification and periosteal differentiation. Below the promontory is the round window niche and the membrana tympani secondaria. Above the promontory is the oval window and stapes. Above the stapes is the facial nerve in its canal. Above the facial nerve is the prominence of the horizontal canal. To the median side of the prominence are the ampullæ of the superior and horizontal canals with their cristæ. Below the ampullæ is the utriculus with its macula. Below the utriculus is the sacculus. Median to the stapes is the cysterna perilymphatica, free of syncytial reticulum. Median to the secondary tympanic membrane and promontory is the beginning of the cochlea. The periotic capsule shows beginning ossification.

Fig. 27. Rat embryo (human age, four months). Horizontal section through the cochlea, vestibular apparatus and tympanum. Resorption of mesenchyme in the tympanum is under way. The stapes has assumed its adult form, but the stapedial artery has not yet disappeared. Note the differentiation between that portion of the labyrinth capsule which has become the footplate and the anterior crus. The writer believes he is the first to show this origin of the stapedial footplate. The annular ligament has formed. The canal of the facial nerve, at this level, shows a deficiency anteriorly. Promontory periosteum is represented by a black line. To the median side of the stapedial footplate is the cysterna, devoid of syncytial reticulum. In the cysterna is a section of sacculus and a portion of the utriculus. Median to the cysterna are sections of the superior and posterior canals. Dorsomesial to the facial nerve is a section of the superior canal. Ventrally the cochlea is viewed. The scalæ are free of syncytial reticulum. Up through the modiolus the cochlear fibers are passing toward the spiral ganglion. The membrana Reisnerri is thin but not yet the delicate structure it will become. The tectorial membrane rests upon the organ of Corti. Rosenthal's canal is

black with cochlear fibers. The ligamentum spiralis and stria vascularis are quite adult in appearance. To the median side of the labyrinth is the brain. We note a definite process of ossification about the cochlea and the posterior and the superior canals.

Fig. 28. Rat embryo (human age, four months). Another view of the same cochlea as in Fig. 27. Here we get a splendid picture of the spiral ganglion and the nerve filled Rosenthal's canal. The sulcus internus, limbus spiralis, tectorial membrane, organ of Corti, etc., can be seen to advantage. Perichondrium-periosteum appears as a black line. The advance of ossification is clearly visible.

Fig. 29. Rat embryo (human age, 4 months). From the same specimen as Fig. 28. A section through an ampulla of a semicircular canal, showing a crista. Note the adult cupola and hairs, the neuroepithelium, the fibers of the nervus utriculo-ampullaris. The membranous ampulla is supported by syncytial reticulum rests. Ossification around the ampulla is well under way.

Fig. 30 A. and B. An adult human labyrinth dissected. From the median side we see the nervus cochlearis and cochlea, the nervus facialis, the nervus vestibularis, the ampullæ of the superior and posterior canals and the crus communis. From the lateral side we note the two and one-half turns of the cochlea, the sacculus and ductus reuniens, the utriculus with the ampullæ of the superior and horizontal canals and the ampulla of the posterior canal. The membranous labyrinth is surrounded by lamellar bone, the periotic capsule.

Fig. 31. A section through an adult human cochlea (slide presented by Prof. Heinrich Neumann), showing the helicotrema (only communication between the scala vestibuli and scala tympani), the organ of Corti with its canal, pillars of Corti and hair cells, the basilar membrane, etc. The membrana tectoria is artificially standing away from the hair cells. On its under surface is Hensen's stripe. This specimen shows pathology (ectasia of cochlear duct, atrophy of ligamentum spiralis, arteriosclerosis of the stria, adhesions between vestibular membrane and basilar membrane of second turn, etc.), which does not concern us here.

Surrounding the cochlea is normal lamellar, periotic bone and numerous so-called cartilaginous rests.

Fig. 32. Human, adult, frontal section through ear, showing tympanum, promontory, footplate of stapes with annular ligament, round window with membrana tympani secondaria, basal turn of cochlea, geniculate ganglion, cysterna perilymphatica, etc.

Fig. 33. Human, adult, section through an ampulla with crista. Note the membranous canal and supporting fibers, nervus utriculo-ampullaris, neuro-epithelium, cupola and hairs.

Fig. 34. Internal auditory meatus of a child. Ventrally we note the tractus spiralis foramenulentus for the passage of the cochlear filaments. Dorsally are the several openings for the vestibular fibers. Superiorly is the canal for the facial nerve.

We are now to consider the development of the external auditory canal, membrana tympani and auricle.

Fig. 35. Domestic duck embryo (human age, four weeks). At the head end of the embryo is seen the frontonasal process, the eye, the first and second branchial arches. Between the mandibular part of the first arch and the second or hyoid arch is the first branchial groove. This groove will deepen to form the external auditory canal. Tubercles will appear on the mandibular and hyoid arches which will eventually form the auricle. Between canal and tympanum, mesenchyme will thin down to form the drum membrane (middle layer). Ectoderm of the canal will cover its outer surface; entoderm of tympanum, its inner surface.

Fig. 36. Norway rat embryo (human age, five weeks). The frontonasal process now shows well developed olfactory pits. The maxillary portion of the first arch has grown larger and the mandibular portion is coming forward, soon to fuse with the arch of the opposite side to form the mandible. The first branchial groove (external canal) is deeper.

Fig. 37. Human embryo, age six weeks. Note the prominence of the upper and lower limbs. On the mandibular and hyoid arches can now be seen the tubercles which will enlarge, then coalesce, to form the auricle.

Fig. 38. Embryo, age seven weeks. The fingers are well developed; the upper limb is much longer than in Fig. 37. Branchial

arches are no longer visible as such. The mandibular-hyoid tubercles have coalesced to form the auricle. The delicate amnion and chorion with its villi are well demonstrated.

Fig. 39. Embryo, age eight weeks. Frontal view, showing the prominence of the auricle, the olfactory openings pointing forward, chorionic villi, etc.

Fig. 40. Rat embryo (human age, 10 weeks). To the right is the external auditory canal with auricle above and below. The periotic capsule is still of cartilage. Syncytial reticulum is thinning, and in several places has disappeared to form periotic spaces. Two ampullæ show cristæ with neuro-epithelium but no hair cells.

Fig. 41. Rat embryo (same specimen as No. 40). We see the drum membrane on the right with its inner entodermal layer and its outer ectodermal covering. The external auditory canal, at this level, has approached the tympanum. In the tympanum, gallert Gewebe is thinning. Ventrally is a section of the tympanum and eustachian tube. The labyrinth capsule is of cartilage. The stapes is represented by Reichert's ring, the adult configuration not yet having developed. The stapedial artery, the reason for the ring shape, is plainly discernible. The typical footplate of stapes, with the annular ligament, has not yet made its appearance. Syncytial reticulum in the cysterna is thinning.

Fig. 42. An adult human ear in frontal section (specimen presented by Prof. Gustav Brühl). The first branchial groove is now a deep canal (external auditory), lined with epidermis which continues over the thinned drum membrane. Inferiorly is the glenoid fossa. In the tympanum we note the processus cochleariformis with the tendon of the tensor tympani muscle, passing outward to the malleus, which articulates above with the incus. The superior ligaments extend between malleus and incus and the tegmen tympani. Above the processus cochleariformis is the geniculate ganglion of the facial nerve. Median to the tympanum is the cochlea. Below the cochlea is the internal carotid artery.

We are now to consider osteogenesis of the periotic capsule.

Fig. 43. Rat embryo (human age, 4 months). According to T. H. Bast, the steps in osteogenesis are: (1) change in the

chemistry of the cartilage cell, resulting in a darker stain; (2) swelling of the cartilage matrix; (3) rupture of perichondrium; (4) wandering in through the rupture, new vessels and connective cells which assume the rôle of osteoblasts; (5) excavation of cartilage by the newly formed vessels; (6) deposit of bone along the perichondrium; (7) entrance of osteoblasts into matrices, destruction of cartilage cells and deposit of bone without loss of matrix form; (8) epithelioid arrangement of osteoblasts around the limits of the excavated areas; (9) deposit of layers of bone (lamellar) about these osteoblasts. In this manner are developed perichondral bone, endochondral bone and interchondral bone. The interchondral bone becomes the lamellar bone of the adult capsule; the endochondral bone, the so-called cartilage rests (in the interglobular spaces).

In the section here presented we note above, a typical rupture of perichondrium. Through the rupture are swarming connective cells and vessels. To the right of the rupture is a dark area of perichondral bone. Below this are a number of cartilage matrices filled each with several osteoblasts which have destroyed the cartilage cells and are converting the cartilage matrices into bone. On the left are large excavated areas lined and more or less filled with osteoblasts producing interchondral or lamellar bone.

Fig. 44. Rat embryo (human, age 4 months), showing early changes in the cartilage. On the left is normal periotic capsule cartilage. In the center is a strip of swollen cartilage matrices. Between the two is a strip of darker staining cartilage cells. To the right are excavated areas lined and filled with osteoblasts. Here and there are matrices filled with osteoblasts.

Fig. 45. Adult human periotic capsule, showing lamellar bone and so-called cartilaginous rests (in interglobular spaces). These, according to Bast, are ossified cartilage matrices.

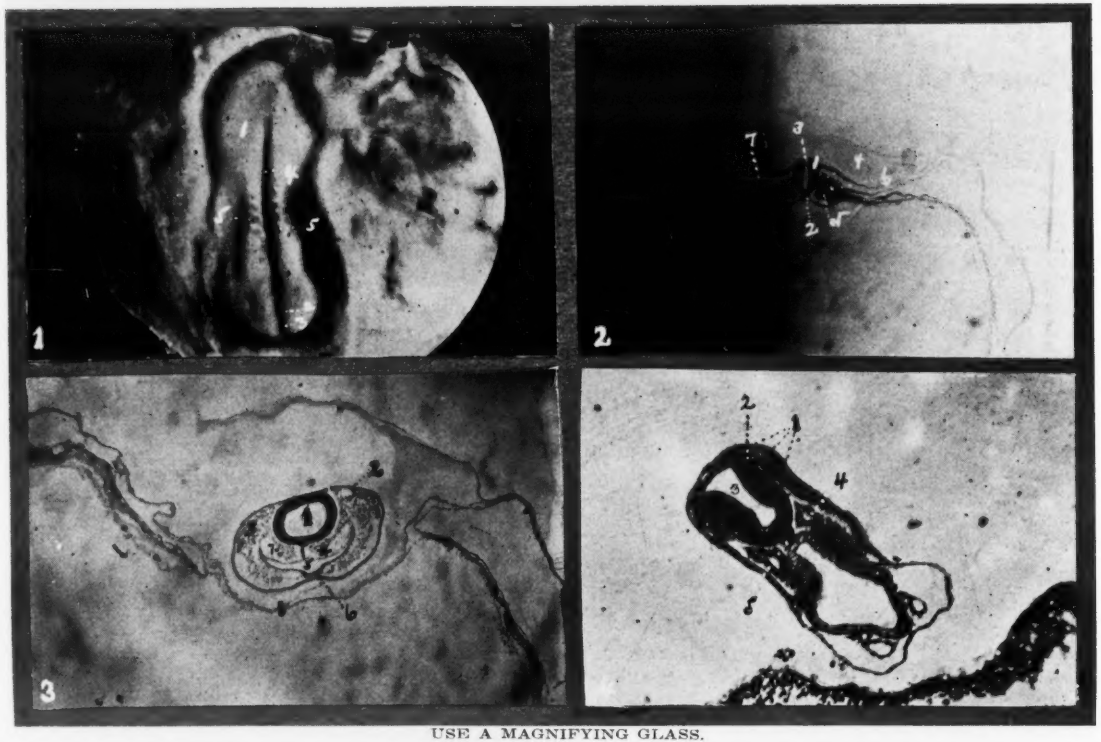
When things go wrong:

Fig. 46. Adult human ear (slide presented by Prof. Heinrich Neumann), showing stapes enmeshed in a network of mesenchymal rests. These bands are soft and elastic and in no way interfere with stapedial movement. If infection occurs in such an ear, these bands become infiltrated and are finally converted

into fibrous tissue which, upon contracting, fixes the stapes almost as completely as does otosclerosis. This explains, I believe, some of the cases of marked impairment of hearing following an ordinary acute middle ear suppuration.

Fig. 47. Adult cochlea of a deafmute (slide presented by Prof. Heinrich Neumann). Lamellar bone with ossified cartilage rests, and in part fiber bone not unlike that of otosclerosis, surround the cochlea. The spiral ganglion is absent, Rosenthal's canal is empty, the basilar membrane is without an organ of Corti, the limbus is low, the ductus cochlearis is ectatic, the scalæ show fiber bone formation and strands of syncytial reticulum. The stria vascularis is present. The myelencephalon failed to throw out ganglion tissue, the auditory vesicle did not differentiate in the usual manner and mesenchymal differentiation proceeded abnormally so that in the production of this case of deafmutism all tissues which participate in the development of the organ of hearing are involved.

CARLETON BUILDING.



USE A MAGNIFYING GLASS.

FIGURE 1.

Rabbit (human age, 10 days)—(1) ectoderm of embryonic disc, (2) neuro-enteric canal, (3) primitive streak, (4) neural groove, (5) segments.

FIGURE 2.

Chicken (human age, 12 days)—(1) neural fold, (2) notochord, (3) neural groove, (4) somatic mesoderm, (5) splanchnic mesoderm, (6) caelom, (7) ectoderm.

FIGURE 3.

Chicken (human age, 16 days)—(1) neural tube, (2) ectoderm, (3) mesenchyme, (4) notochord, (5) pharynx, (6) pharyngeal membrane, (7) descending aorta, (8) entoderm and ectoderm of proamnion.

FIGURE 4.

Rat (human age, 20 days)—(1) auditory placode, (2) acoustic ganglion, (3) myelencephalon, (4) caelom, (5) notochord.

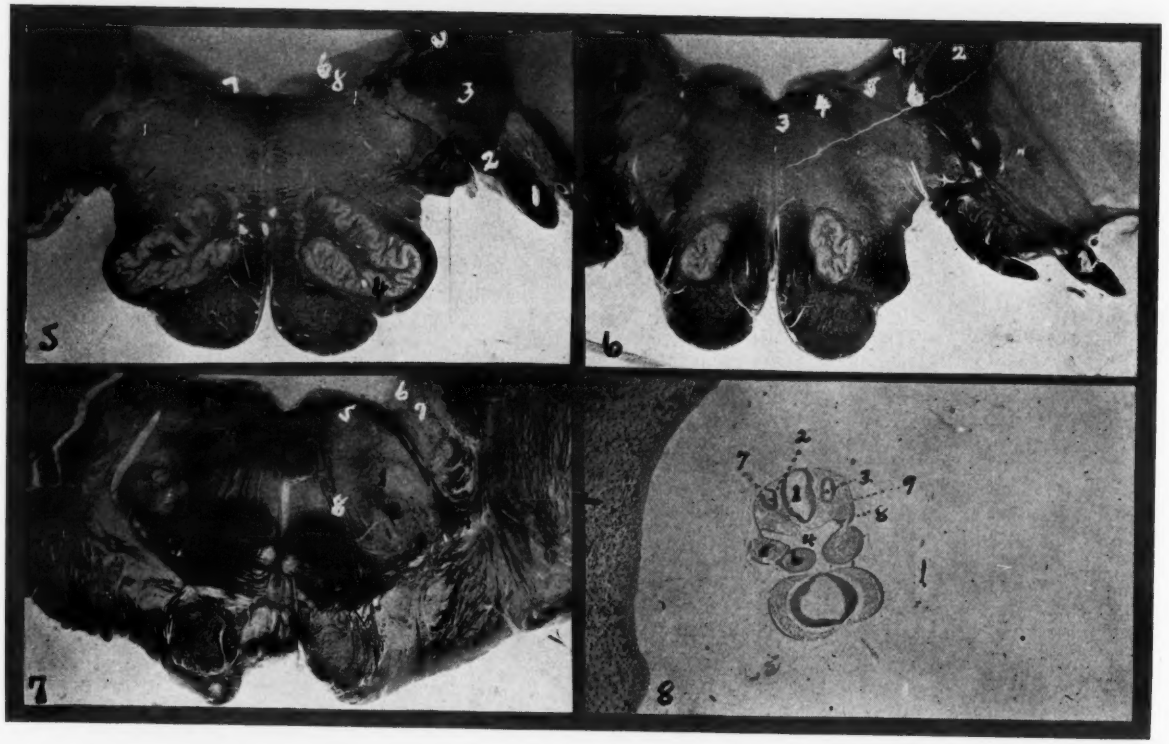

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FIGURE 5.

Adult human medulla (specimen presented by Prof. Max Bielschowsky) — (1) ramus cochlearis, (2) anterior cochlearis nucleus, (3) corpus restiforme, (4) inferior olive, (5) tuberculum acusticum, (6) striæ acusticæ, (7) nucleus eminentiæ teretis, (8) nucleus vestibularis parvicellularis, (9) descending fibers of the vestibular, (10) tractus nucleocerebellaris, (11) Deiter's nucleus.

FIGURE 6.

Adult human medulla (higher level than Fig. 5). (Specimen presented by Prof. Max Bielschowsky.)—(1) ramus vestibularis, (2) restiforme fibers, (3) fasciculus longitudinalis posterior, (4) nucleus of abducens, (5) small cell triangular vestibular nucleus, (6) Deiter's nucleus, (7) nucleus angularis.

FIGURE 7.

Adult human pons (specimen presented by Prof. Max Bielschowsky)—(1) pedicle fibers, (2) superior olive, (3) corpus trapezoides, (4) facial, (5) abducens nucleus, (6) small cell vestibular nucleus, (7) Deiter's nucleus, (8) abducens.

FIGURE 8.

Rat (human age, 24 days)—(1) myelencephalon, (2) foveola labyrinthica, (3) auditory vesicle, (4) pharynx, (5) maxillary division of first arch, (6) mandibular division of first arch, (7) appertura vesiculæ auditivæ, (8) entoderm, (9) ectoderm.

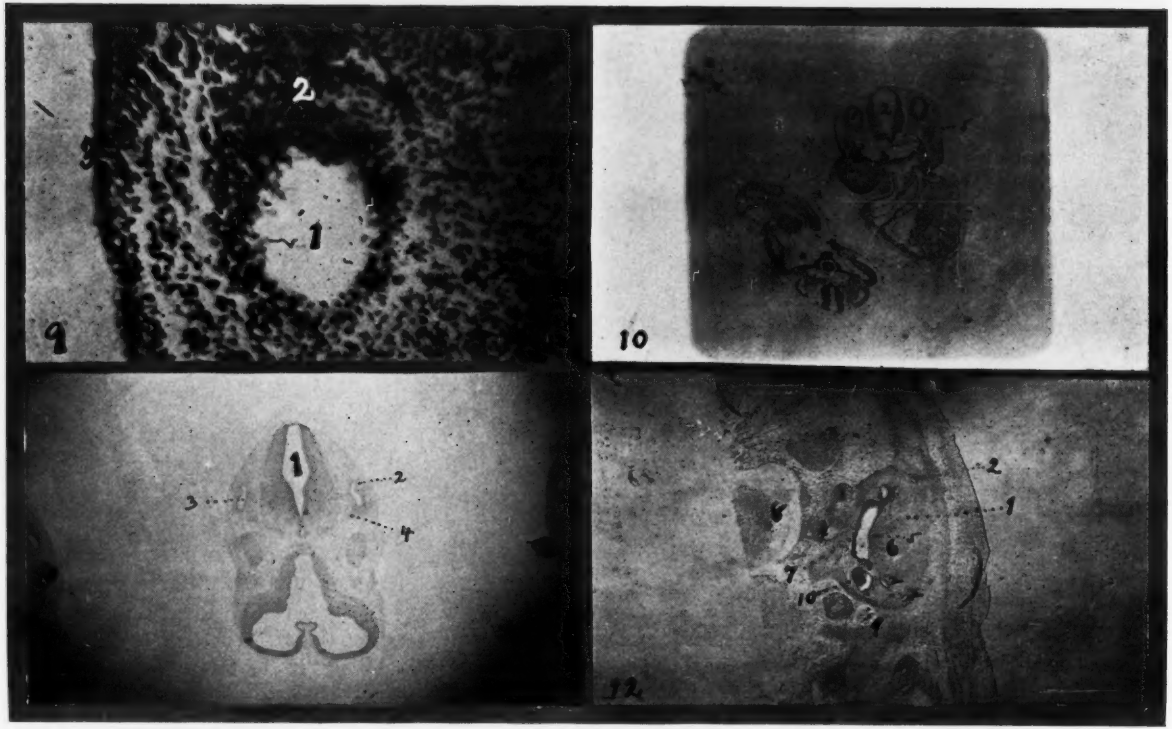
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FIGURE 9.

Human, 28 days—(1) auditory vesicle, (2) condensed mesenchymal cells, (3) ectoderm.

FIGURE 10.

Rat (human age, 28 days)—(1) auditory vesicle, (2) myelencephalon, (3) pharynx, (4) mesenchyme, (5) ectoderm.

FIGURE 11.

Rat (human age, 31 days)—(1) myelencephalon, (2) recessus labyrinthi, (3) auditory vesicle, (4) acoustic ganglion.

FIGURE 12.

Rat (human age, 45 days)—(1) precartilage, (2) semicircular canal, (3) vestibular ganglion, (4) facial nerve, (5) constriction for division of vesicle into utriculus and sacculus, (6) sacculus, (7) thickened epithelium for maculæ, (8) myelencephalon, (9) ductus cochlearis, (10) spiral ganglion.

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FIGURE 13.

Rat (human age, 45 days)—(1) semicircular canal showing thickened epithelium for crista ampullaris, (2) condensed mesenchyme.

FIGURE 14.

Human labyrinth, 60 days—(1) cochlea, (2) precartilage, (3) halo of beginning dedifferentiation of precartilage into syncytial reticulum (Arbeit of George Streeter).

FIGURE 15.

Human labyrinth, 60 days—(1) thickening of epithelium for crista ampullaris, (2) halo of dedifferentiation, (3) precartilage, (4) Scarpa's ganglion, (5) ramus utriculo-ampullaris of vestibular nerve.

FIGURE 16.

Rat (human age, 65 days)—(1) precartilage, (2) thickening of ductus cochlearis wall to form organ of Corti, etc., (3) stapedial ring, (4) artefact.

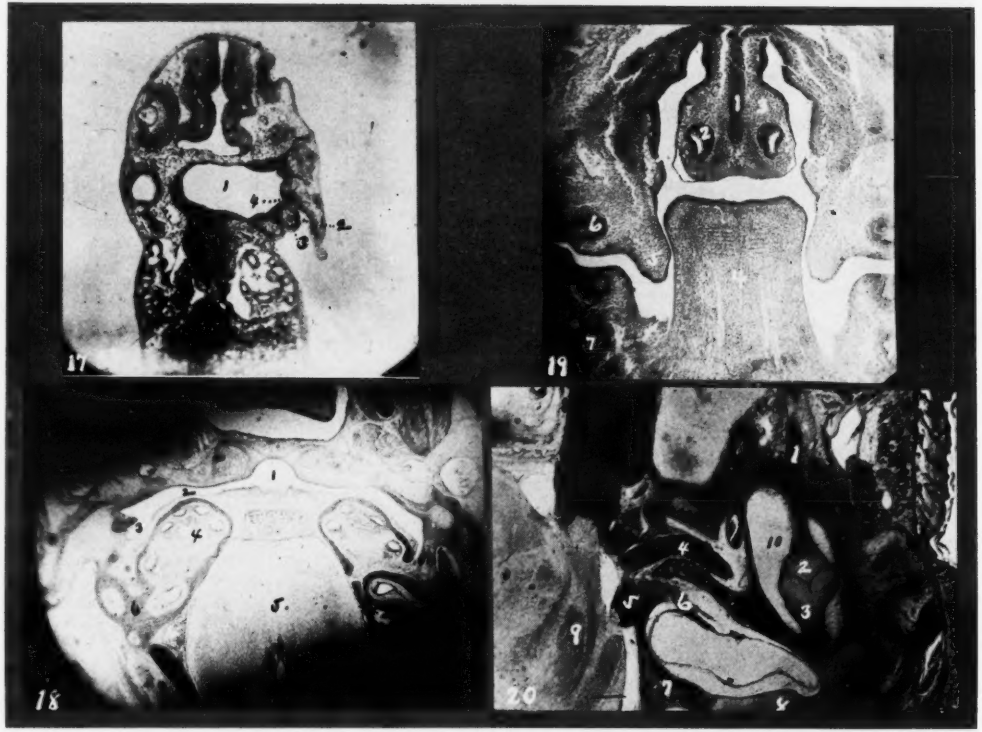
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FIGURE 17.

Tadpole — (1) pharynx, (2) branchial groove, (3) branchial arch, (4) pharyngeal pouch.

FIGURE 18.

Rat (human age, 3 months)—(1) pharynx, (2) first pharyngeal pouch (eustachian tube), (3) tympanum, (4) cochlea, (5) myelencephalon, (6) vestibular labyrinth.

FIGURE 19.

Rat (human age, 2 months)—(1) precartilage of nasal septum, (2) vomeronasal organ of Jacobson, (3) mesenchyme, (4) tongue, (5) lateral palatine process of maxillary division of first arch, (6) enamel organ, (7) Meckel's cartilage.

FIGURE 20.

Rat (human age, 4 months)—(1) Meckel's cartilage, (2) malleus, (3) incus, (4) facial nerve, (5) Scarpa's ganglion, (6) nervus utriculo-ampullaris, (7) ossification, (8) cartilage, (9) myelencephalon, (10) tympanum.

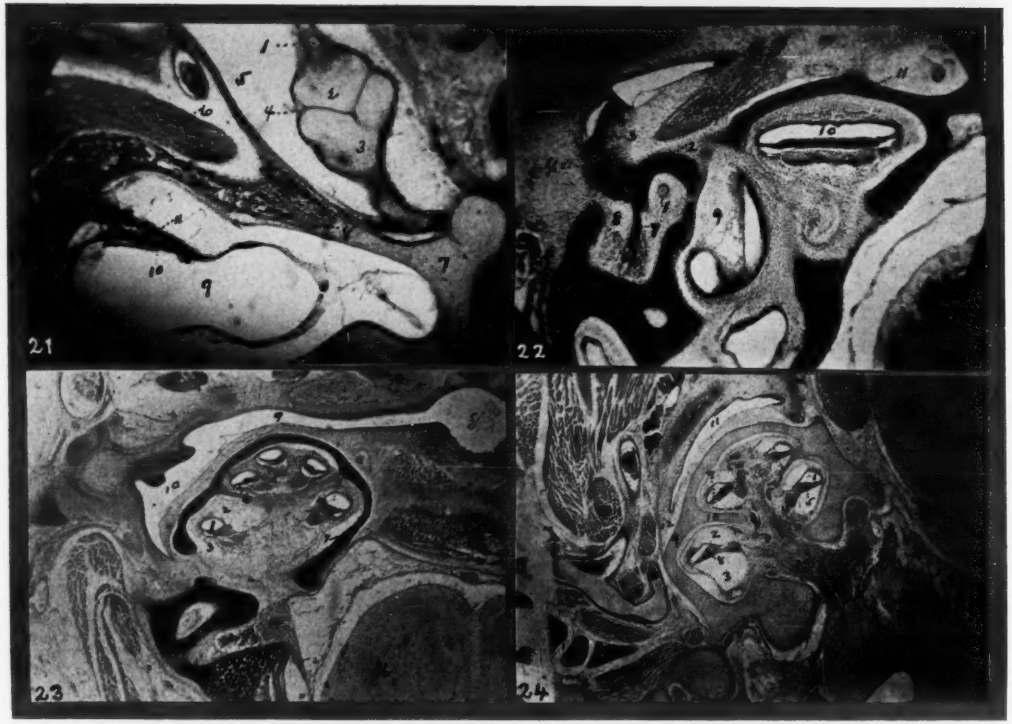
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FIGURE 21.

Rat (human age, 4 months) (1) vacuole in Meckel's cartilage, (2) malleus, (3) incus, (4) beginning joint formation, (5) tympanum, (6) facial nerve, (7) cartilage, (8) ossification, (9) utriculus, (10), macula (11), ramus utriculo-ampullaris.

FIGURE 22.

Rat (human age, 3 months)—(1) stapedial artery, (2) stapedial ring, (3) remains of Reichert's cartilage, (4) atrophy of Reichert's cartilage, (5) tensor tympani muscle, (6) stapedial muscle, (7) tendon of stapedial muscle, (8) mesenchyme in tympanum, (9) cysterna perilymphatica showing resorption of syncytial reticulum, (10) ductus cochlearis, (11) cartilaginous periotic capsule.

FIGURE 23.

Rat (human age, 3 months)—(1) ductus cochlearis, (2) resorption of syncytial reticulum in the scala vestibuli, (3) scala tympani, (4) spiral ganglion, (5) inner greater thickening for inner hair cells, limbus spiralis, etc., (6) outer lesser thickening for outer cells of Corti, (7) cartilaginous periotic capsule, (8) pharynx, (9) eustachian tube, (10) tympanum, (11) membrana tectoria, (12) malleus, (13) drum membrane, (14) Scarpa's ganglion, (15) nervus vestibularis, (16) myelencephalon.

FIGURE 24.

Rat (human age, 3½ months)—(1) ductus cochlearis, (2) scala vestibuli, (3) scala tympani, (4) spiral ganglion, (5) Rosenthal's canal, (6) limbus spiralis, (7) canal of Corti, (8) sulcus internus, (9) cartilaginous periotic capsule, (10) cartilage from capsule forming modiolus, (11) tympanum, (12) mesenchyme in potential tympanum, (13) cochlearis fibers in modiolus.

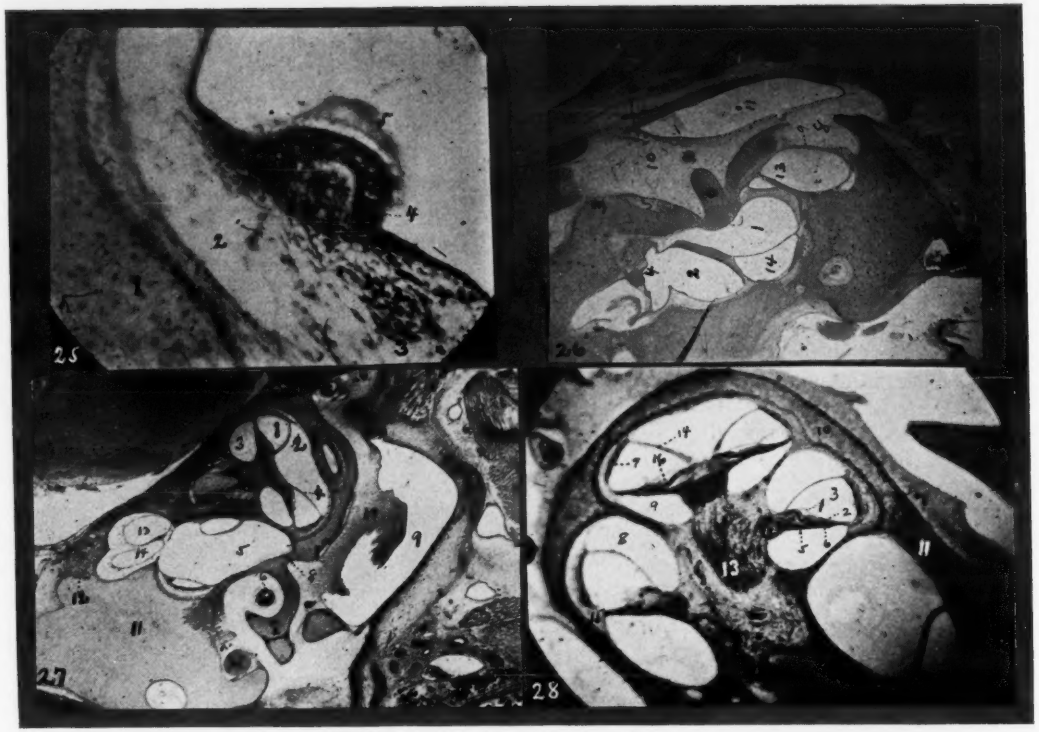
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FIGURE 25.

Rat (human age, 3½ months)—(1) periotic capsule of cartilage, (2) periotic space of ampulla, (3) fibers of ramus utriculo-ampullaris, (4) neuro-epithelium of crista, (5) hairs and cupola.

FIGURE 26.

Rat (human age, 3¾ months)—(1) cysterna perilymphatica, (2) utriculus, (3) prominence of horizontal canal, (4) cristæ of superior and horizontal canals, (5) facial canal, (6) stapes, (7) promontory, (8) niche of round window, (9) membrana tympani secondaria, (10) mesenchyme, (11) tympanum, (12) periotic capsule showing beginning ossification, (13) scala tympani, (14) sacculus.

FIGURE 27.

Rat (human age, 4 months)—(1) ductus cochlearis, (2) scala vestibuli, (3) scala tympani, (4) vestibular (Reisnerri) membrane, (5) cysterna perilymphatica, (6) stapedial artery, (7) stapes, (8) annular ligament, (9) tympanum, (10) mesenchyme, (11) cartilage of periotic capsule, (12) ossification in capsule, (13) superior canal, (14) posterior canal, (15) facial nerve.

FIGURE 28.

Rat (human age, 4 months)—(1) membrana tectoria, (2) organ of Corti, (3) ductus cochlearis, (4) limbus spiralis, (5) Rosenthal's canal, (6) basilar membrane, (7) stria vascularis, (8) scala vestibuli, (9) scala tympani, (10) cartilage of periotic capsule, (11) bone of periotic capsule, (12) spiral ganglion, (13) modiolus with cochlear fibers, (14) membrana Reisnerri, (15) ligamentum spiralis, (16) sulcus internus.

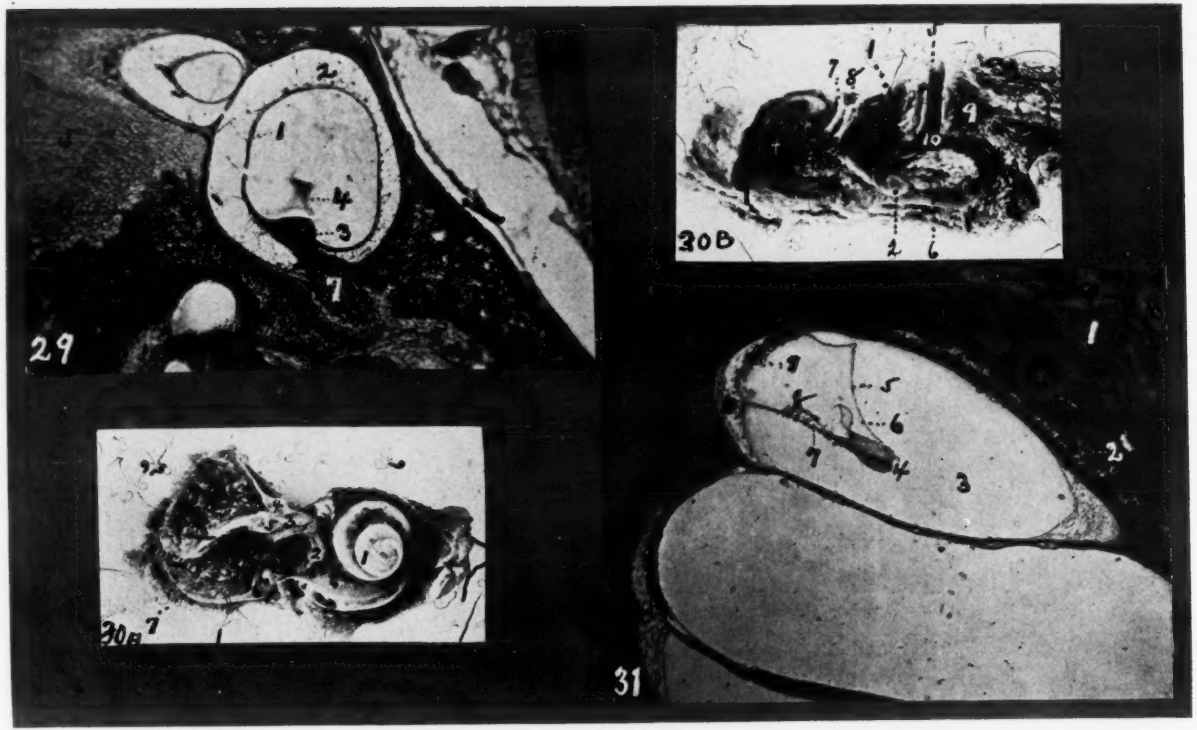
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FIGURE 29.

Rat (human age, 4 months)—(1) membranous ampulla, (2) perilymphatic space with supporting fibers, (3) neuro-epithelium of crista, (4) hairs, (5) cartilage of periotic capsule, (6) bone of capsule, (7) ramus utriculoampullaris.

FIGURE 30A.

Human labyrinth, adult, external view—(1) cochlea, (2) sacculus, (3) utriculus (4) ampulla of superior canal, (5) ampulla of horizontal canal, (6) ampulla of posterior canal, (7) periotic capsule of bone.

FIGURE 30B.

Human labyrinth, adult, median view—(1) ampulla of superior canal, (2) ampulla of posterior canal, (3) ramus cochlearis, (4) cochlear, (5) superior canal, (6) posterior canal, (7) facial nerve, (8) vestibular nerve, (9) periotic capsule, (10) crus communis.

FIGURE 31.

Adult human cochlea (slide presented by Prof. Heinrich Neumann)—(1) lamellar bone of capsule, (2) cartilaginous rests (ossified matrices), (3) helicotrema, (4) hamulus, (5) membrana Reisnerri, (6) membrana tectoria, (7) basilar membrane, (8) organ of Corti, (9) stria vascularis, (10) ligamentum spiralis.

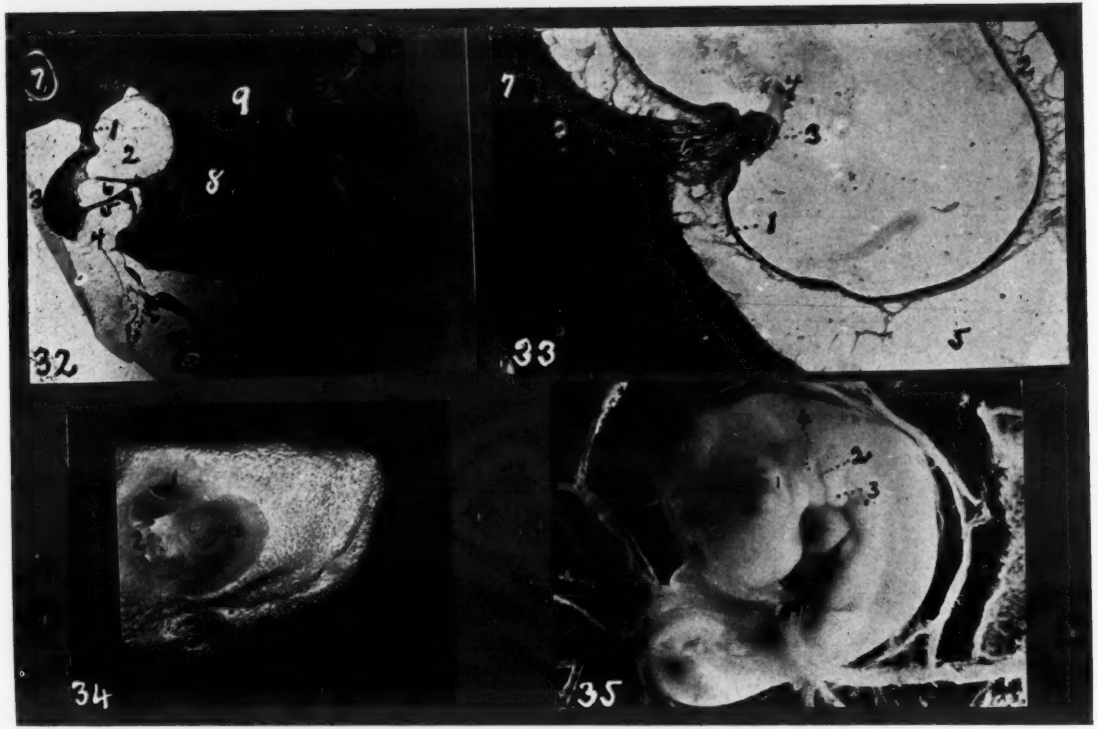
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FIGURE 32.

Adult human—(1) annular ligament, (2) cysterna perilymphatica, (3) promontory, (4) niche of round window, (5) membrana tympani secondaria, (6) basal turn of cochlea (scala tympani), (7) geniculate ganglion, (8) cartilaginous rests, (9) lamellar bone.

FIGURE 33.

Adult human — (1) membranous ampulla, (2) perilymphatic space with syncytial reticulum rests (supporting fibers), (3) neuro-epithelium of crista, (4) hairs and cupola, (5) perilymphatic space, (6) fibers of utriculo-ampullaris nerve, (7) same on cross section.

FIGURE 34.

Internal auditory meatus of a child—(1) facial canal, (2) openings for vestibular nerve fibers, (3) tractus spiralis foramenulentus.

FIGURE 35.

Domestic duck (human age, 4 weeks)—(1) eye, (2) first branchial groove (external auditory canal), (3) second or hyoid arch, (4) mandibular division of first arch.

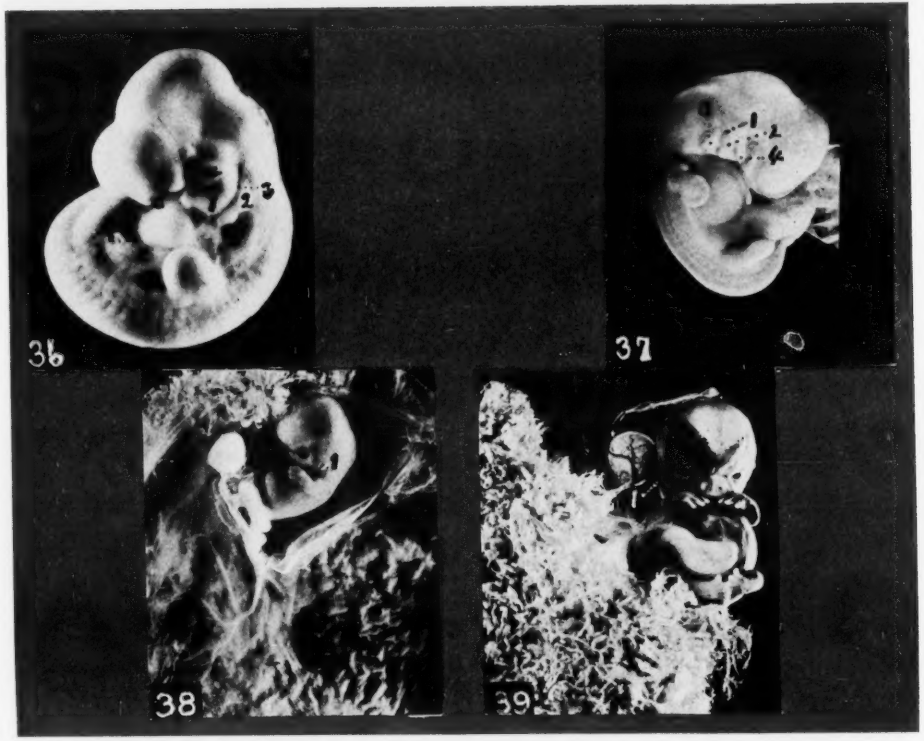
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FIGURE 36.

Norway rat (human age, 5 weeks)—(1) mandibular arch, (2) hyoid arch, (3) first branchial groove, (4) maxillary division of first arch; (5) olfactory pit.

FIGURE 37.

Human, six weeks—(1) first branchial groove, (2) mandibular arch showing tubercles for auricle, (3) hyoid arch showing tubercles for auricle, (4) maxillary arch.

FIGURE 38

Embryo, seven weeks—(1) primitive auricle (tubercles fused).

FIGURE 39.

Embryo, eight weeks—(1) auricle.

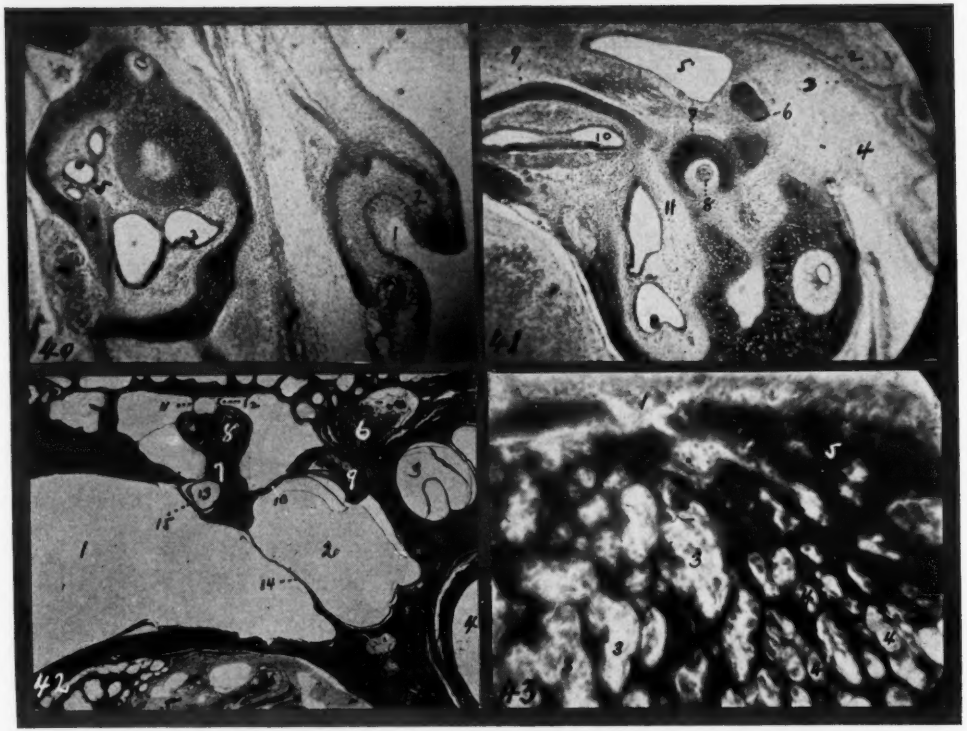
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FIGURE 40.

Rat (human age, 10 weeks)—(1) external auditory canal (first groove), (2) auricle, (3) ampulla with hairless crista, (4) periotic cartilage, (5) syncytial reticulum disappearing.

FIGURE 41.

Rat (human age, 10 weeks)—(1) external auditory canal, (2) ectodermal layer of drum membrane, (3) entodermal layer of drum membrane, (4) gallert Gewebe in tympanum, (5) tympanum, (6) Reichert's cartilage, (7) stapedial ring, (8) stapedial artery, (9) periotic cartilage, (10) cochlea, (11) syncytial reticulum of cysterna, resorbing.

FIGURE 42.

Adult human (specimen presented by Prof. Gustav Brühl)—(1) external auditory canal, (2) tympanum, (3) cochlea, (4) internal carotid artery, (5) glenoid fossa, (6) geniculate ganglion, (7) malleus, (8) incus, (9) processus cochleariformis, (10) tendon of tensor tympani, (11) superior ligament of malleus, (12) superior ligament of incus, (13) Prussack's space, (14) membrana tympani, (15) Shrapnell's membrane.

FIGURE 43.

Rat (human age, 4 months)—(1) rupture in perichondrium, (2) perichondrium, (3) excavated area lined with osteoblasts, forming interchondral bone, (4) calcification of cartilage matrices (endochondral bone), (5) perichondral bone (Arbeit of T. H. Bast).

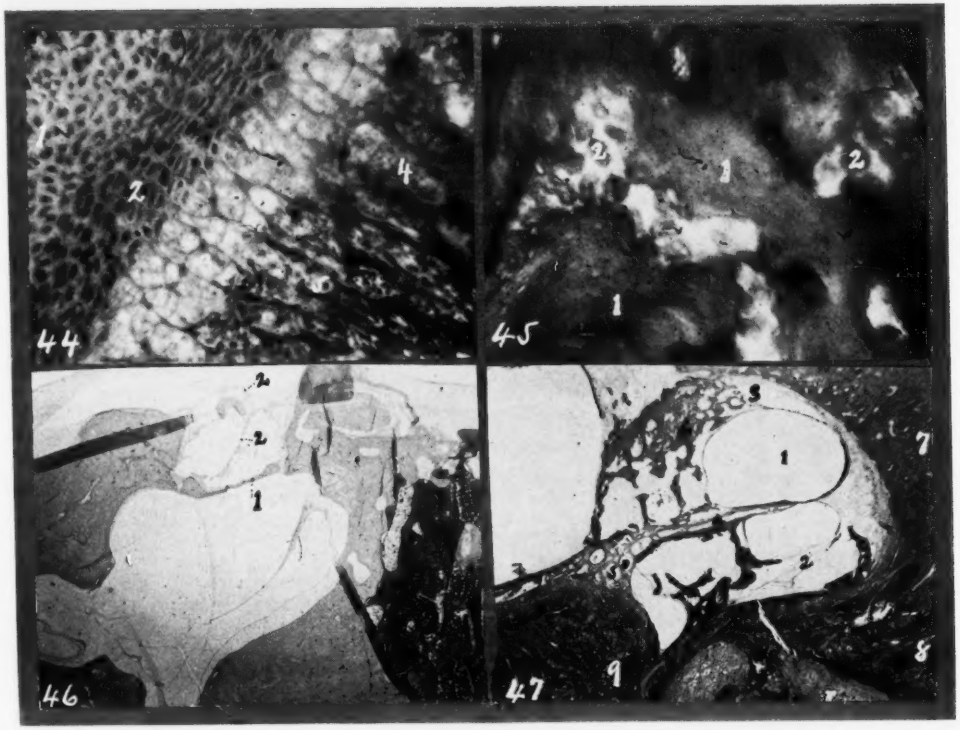
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FIGURE 44.

Rat (human age, 4 months)—(1) cartilage, (2) changed cartilage, staining more deeply, (3) swollen matrices, (4) excavation lined and filled with osteoblasts, (5) calcification of matrix (Arbeit of T. H. Bast).

FIGURE 45.

Adult, human periotic capsule—(1) lamellar (interchondral bone, Bast), (2) cartilaginous rests in interglobular spaces (endochondral bone, Bast).

FIGURE 46.

Human, adult (section presented by Prof. Heinrich Neumann)—(1) footplate of stapes, (2) mesenchymal band.

FIGURE 47.

Human, adult, deafmutism (section presented by Prof. Heinrich Neumann)—(1) ductus cochlearis, (2) syncytial reticulum rests in scala tympani, (3) same in scala vestibuli, (4) Rosenthal's canal (empty), (5) area for spiral ganglion (empty), (6) fiber bone in both scalæ and in the aqueductus cochlearis, (7) abnormal periotic capsule bone (resembling otosclerosis), (8) lamellar bone, (9) cartilaginous rests.

LXIX.

THE EFFECT OF TRYPAN BLUE ON EOSINOPHILIA EXPERIMENTALLY PRODUCED IN THE MAXILLARY SINUS MUCOSA.

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The presence of a large number of eosinophiles in the mucosa of the nasal cavities has been definitely associated with allergy; in fact, modern workers in allergy look upon eosinophilia in the nasal mucosa as a sign of allergy. That this tissue eosinophilia may or may not be associated with an eosinophilia of the blood stream has been shown by Hansel,[1] who gives a review of the literature, and also personal observations on allergy and its relation to the nasal mucosa. Mayr and Montcorps[2] point out that the spleen seems to be related in some unexplained way to eosinophilia and the allergic diathesis. They state that with an increased activity of the spleen as in malaria, where the spleen and reticulo-endothelial system in general have to take care of an increased amount of products of phagocytic activity, cases were observed where hypo-eosinophilia, or even aneosinophilia were present. They found, also, that the injection of protein free splenic extract caused a rise in red cell count and a relative and absolute decrease in the number of eosinophiles. In cases of allergic dermatitis with eosinophilia, injection of protein free splenic extract caused a fall in the number of eosinophiles and an alleviation of the dermatitis in a certain number of cases.

The phenomenon of the anaphylactic reaction has been of great interest. Formerly it was thought that it took place in the circulating blood stream, but in more recent years, work of Doerr[3] and others indicate that this reaction takes place in the lining of the capillaries—that is, the reticulo-endothelial system. The effect

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of "endothelial blockade" on the anaphylactic reaction by means of colloids, such as the vital dyes (trypan blue, congo red), india ink and other substances, has been the subject of intensive study by immunologists. In 1926, Hans Meyer[4] was able to diminish the extent of anaphylactic shock in various species of experimental animals by means of endothelial blockade or splenectomy, or by a combination of both. These procedures are not uniformly effective, for the results reported by various investigators are conflicting. In a recent paper, Jungeblut and Newnan[5] claim to obtain only a retardation of the death of guinea pigs from experimental anaphylaxis by means of the injection of trypan blue. It seems to be the consensus of opinion that even if the shock of anaphylaxis is hindered by endothelial blockade there is no diminution in the development of antibodies. The factors involved in the related phenomena of allergy and anaphylaxis tend to link the allergic diathesis with the spleen and reticulo-endothelial system in general.

An intense infiltration of eosinophiles of the maxillary sinus mucosa of rabbits by chemical means is reported by Wenner[6] and Wenner and Nemours.[7] This was obtained by irrigating the sinus with calcium combining substances, such as alizarin and sodium citrate. A condition of alkalosis greatly augmented the effect of the calcium combining substance, whereas acidosis inhibited it. We thought that it would be of interest to study the effect of irrigation of calcium combining substance on the maxillary sinus mucosa of rabbits following the introduction of trypan blue into the blood stream. It seemed reasonable to assume that an endothelial blockade produced by trypan blue might affect the production of eosinophilia in the nasal mucous membrane.

EXPERIMENT I.

Rabbits under eight months of age were used as experimental animals. Two control rabbits were injected five times intravenously on alternate days with 10 cc. of a 1 per cent trypan blue solution. On the tenth day they were killed and the maxillary sinus mucosa removed. Formalin fixation was used and paraffin sections four micra thick were prepared. Both hematoxylin and

eosin, and Giemsa methods of staining were employed. The epithelium was found to be apparently normal. No dye was seen within the cells, but a small amount was observed in the capillaries and lymphatics of the basement membrane. Trypan blue is evidently present in suspension in the body fluids, for the appearance of the specimen in the paraffin block is more blue than could be accounted for by the amount of dye in the capillaries and lymphatics.

In a study of the reaction of the chick embryo to trypan blue Latta and Busby[5] conclude that there are not a sufficient number of stained cells present to account for the gross blue color. They give, also, a review of the current opinion as to the pharmacologic effect of trypan blue, and state that trypan blue and some of the other acid azo dyes produce little or no toxic effect on animals but that some investigators have suggested that the cellular and tissue reactions to trypan blue are similar, although much less in degree, to those produced by bacterial invasion of inoculation.

EXPERIMENT II.

Two control rabbits had nasal irrigations of 6-8 cc. of a 1 per cent trypan blue solution on alternate days five times. On the tenth day they were killed. The maxillary sinus mucosa appeared normal with both hematoxylin-eosin and Giemsa stains.

The two preceding experiments demonstrate that trypan blue has no histologic effect on ciliated columnar epithelium .

EXPERIMENT III.

Three rabbits had nasal irrigations of 6-8 cc. of a mixture of 1 per cent trypan blue and .5 per cent sodium alizarin sulphonate on alternate days and were autopsied on the tenth day. In one rabbit the epithelium appeared normal, while in the other two there was a neutrophilic invasion of the epithelium and a slight eosinophilia in the glandular layer. The cilia appeared normal in all cases. The effect of alizarin alone on the mucosa of rabbits is different. Wenner[6] found an invasion of eosinophiles in the basement membrane of the maxillary sinus mucosa following irrigations with calcium combining substances. There also was a

loss of cilia in spots. The normal incidence of eosinophiles in the normal nasal mucous membranes of the rabbit is small, for one has to look over many fields before finding one eosinophile. The results of a combined trypan blue-alizarin solution show that trypan blue has a modifying effect on the action of alizarin; there is no destruction of cilia, and eosinophiles in the tissue are greatly reduced or may be entirely absent. The occurrence of neutrophiles in the epithelium of several of the rabbits may be accounted for by the frequent incidence of nasal infections in rabbits, and also by the introduction of bacteria in the irrigating fluids.

EXPERIMENT IV.

Five rabbits were injected intravenously five times on alternate days with 10 cc. of 1 per cent trypan blue solution. These injections were followed in fifteen minutes with nasal irrigations of 6-8 cc. of .5 per cent alizarin. The rabbits were killed on the tenth day and the maxillary sinus mucosa sectioned. Eosinophilia was absent or only slightly present, as may be seen from the following table:

TABLE I.

Rabbit	Cilia	Eosinophiles	Neutrophiles
125	normal	none	none
50	"	"	moderate in epithelium
51	"	slight in epithelium	none
52	"	"	"
126	"	"	"

The results as indicated in the above table are in marked contrast in regard to eosinophilia and action on cilia to that of normal rabbits irrigated with alizarin solution as reported by Wenner.[6]

EXPERIMENT V.

Four rabbits were treated as follows: On the first and third day they were injected intravenously with 10 cc. of a 1 per cent trypan blue solution. On the second, fourth, sixth, eighth and tenth day about 8 cc. of blood was withdrawn by cardiac punc-

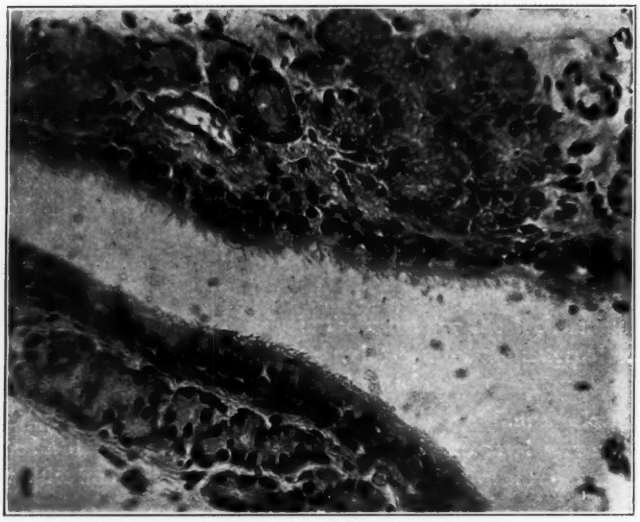

Fig. 1. Rabbit 50—Intravenous injections of trypan blue, and alizarin irrigations to nose. Epithelium is apparently normal, no cellular infiltration.

ture in a luer syringe containing mineral oil (to prevent loss of CO_2). Then two grams of sodium bicarbonate were injected intravenously as a 5 per cent solution. In ten minutes blood was again taken by cardiac puncture and the pH and CO_2 content were determined.

After the second cardiac puncture, the nose was irrigated with 6-8 cc. of a .5 per cent sodium alizarin sulphonate solution. On the eleventh day the rabbits were killed and examined, and sections made of the maxillary sinus mucosa. The number of trypan blue injection was limited to two, for it is excreted slowly; even on the tenth day the blood plasma was tinged blue, showing that throughout the experiment trypan blue was present to exert any possible effect on the reticuloendothelial system. The effect of the injection of sodium bicarbonate can be seen in table 2.

TABLE II.

Number of Animal and Date	Amt. of $NaCO_3$ injected intravenously	pH of blood before injection at room temp.	pH of blood 10 min. after injection at room temp.	CO_2 content of blood after injection. Vols. per c't	CO_2 content of blood after injection. Vols. per c't
5-19-31 No. 113	2 grams	7.40	7.40	43.0	65.5
5-21-31 No. 113	2 grams	7.30	7.70	61.3	93.7
5-23-31 No. 113	2 grams	7.54	7.83	42.7	77.6
5-25-31 No. 113	2 grams	7.26	7.75	40.0	78.7
5-27-31 No. 113	2 grams	7.54	7.84	42.3	85.0

Results: Moderate number of neutrophiles in epithelium; also moderate number of eosinophiles.

The results of this experiment are presented in table 3. As can be seen in table 3, marked eosinophilia was obtained in only one case, whereas in rabbits not injected with trypan blue eosinophilia is found in every case.

TABLE III.

Rabbit	Cilia	Eosinophiles	Neutrophiles
106	normal	none	moderate in epithelium
111	"	"	" " "
113	"	moderate in epithelium and submucosa	" " "
116	"	marked number in epithelium and submucosa	slight in epithelium

DISCUSSION.

From the results of the above experiments it seems that trypan blue has an inhibiting effect on the production of experimental eosinophilia in sinus mucous membrane of rabbits. From the results of the work of Mayr and Montcorps it would seem that there is an intimate relationship between the reticulo-endothelial system (capillary endothelium of bone marrow, lymph sinuses, splenic veins and adrenal capillaries; see Kaufmann[2]), and eosinophilia, consequently anything that inhibits the function of the reticulo-endothelial system, such as accomplished by colloidal blockade, may inhibit the production of eosinophilia.

CONCLUSIONS.

1. Trypan blue inhibits the production of experimental eosinophilia in nasal sinus mucous membrane by means of a blockade of the reticulo-endothelial system.

2. Trypan blue has a modifying effect on the action of calcium-combining substances on the cilia of the maxillary sinus mucous membrane.

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LXX.

AMATEUR CINEMATOGRAPHY IN TECHNICAL TEACHING.*

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Although motion pictures have long been utilized for the demonstration of technical subjects, the expense of standard film and standard apparatus and the technical complications involved in the making of good pictures have restricted their use to subjects whose general popularity insures a distribution which justifies the expense.

However, the amateur cinematographer now has at his disposal effective and relatively inexpensive equipment for an endless variety of teaching material, peculiarly adapted to the demonstration of highly technical subjects. The comparatively recent perfecting of substandard apparatus and high speed reversible film, together with the active interest which the College of Surgeons and the American Medical Association are displaying, furnish a fresh incentive for the development of this type of work in medical demonstration.

The controversy in regard to the choice of standard 35 mm. or amateur 16 mm. film seems to me pointless. Each has its advantages—in fact, each can accomplish certain things which the other cannot. The 16 mm. film has been subjected to unreasonable and unscientific comparisons with the standard film and has been unjustly criticised. A frame of the small film transmits approximately one-fourth as much light as the standard size. When these two are projected side by side at the same distance from the screen the smaller may suffer by comparison.

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*From the Department of Otolaryngology, Washington University School of Medicine, St. Louis. Presented as a motion picture before the Middle Section, American Laryngological, Rhinological and Otological Society, Omaha, January, 1931.

The silver granules in the emulsion are *relatively* larger in the smaller film and hence are more noticeable when the small projector is called upon to cover large screen areas.

On the other hand, the reversal process permits the film which ran through the camera to be redeveloped from a negative into a positive and used for projection, thus cutting film stock and developing expense in half.

To put it plainly, finished standard film costs four times as much to produce as 16 mm. reversible—an expense which ordinary classroom requirements do not justify.

The photographing of operations comes first to mind, but it is by no means the only application nor even the most important. There are several other uses to which motion pictures can be put in the daily routine of medical teaching and in the presentation of new methods before scientific societies. Descriptions of complicated methods and those requiring special apparatus are purposely avoided here. With the exception of cinemicrography, the work outlined may be accomplished by means of an ordinary amateur motion picture camera, a good tripod, 200 to 500 watts of illumination and an exposure meter. Arc lamps are made which are adapted to amateur purposes and are unusually efficient, but they are not recommended for use in the operating room, and since the advent of superspeed film are unnecessary.

Operations.—In introducing the camera into the operating room, the apparatus must be so arranged as to obtain an adequate and well illuminated field without inconveniencing the surgeon. There must be an unobstructed space between the lens and the field at all times, and the camera must rest on a firm tripod. The expedient of holding it in the hand and dodging about with it to obtain whatever views may present themselves is unsatisfactory both as to illumination and focusing, and the resulting picture is confusing.

Various supports have been devised to hold and wind the camera, which is fixed in an advantageous position and focused upon the converging point of the rays from two or more lamps. These apparatuses, while exceedingly useful, entail an expense which is justified only by frequent and prolonged use. Most

amateur photographers have not a sufficient number of subjects to warrant it.

The camera for ordinary shots should be somewhat above the operative field and to one side, the distance varying according to the nature of the record, from six or eight inches, which is sometimes employed in eye work, to six or seven feet. The field should have as much illumination as possible from sources other than the immediate lamps—that is, from skylight or operating lamps, for two reasons: First, because light from a single source makes a flat picture in which the high lights are strongly illuminated and the shadows are without detail; second, because the depth of focus (depth of field) increases as the iris diaphragm is stopped down, which can be done in direct proportion to the amount of light on the object. This phenomenon is characteristic of all lenses, regardless of their speed. The lamp which constitutes the chief source of light should be placed, ordinarily, about 45° to one side of the camera. The direct focusing device now embodied in several cameras is of great assistance. The exposure meter is invaluable.

Lighting from a single source, though not to be generally recommended, produces harsh contrasts, which in capable hands are sometimes very effective in eliminating confusing detail.

In sighting the field, it should be remembered that the finder is set to one side or above the lens, and allowance should be made for this parallax if the working distance is less than three feet.

Ordinarily the normal speed of 16 frames per second is recommended. If the manipulation is extremely rapid and it is desired to slow down the resulting picture, 24 or 32 frames may be exposed with a corresponding increase in the diaphragm opening or the illumination. If the illumination is insufficient for the largest stop of the lens or if it is desired to speed up the action, a speed of 8 frames may be used, but in this case a jerkiness of action may result.

Unless otherwise indicated by the nature of the demonstration, the camera should be operated intermittently and only when something of interest occurs—i. e., the incision and the special features of the operation. It is unnecessary, tedious and expensive to keep the camera running while, for instance, arteries are being

clamped and ligatures tied. Keep the camera still. Let the motion go on in the field. If the camera is moved about, the result is disturbing.

If sudden close-ups are desired in the course of the operation, it is, of course, possible to readjust the entire camera and tripod, resetting the focus and the diaphragm, but this is cumbersome, leads to technical errors, and is apt to discommode the surgeon. A much better method is to shift from the ordinary lens to one of a longer focus without moving the camera. The two-inch lens, for example, takes in a field one-half as wide and one-half as high as the standard one-inch lens, which means a 2× magnification, one usually sufficient for this type of close-up. The area now is only one-fourth what it was. By changing lenses, the alternations between close-ups and medium shots can be effected at will without inconvenience and without changing the angle of view. In determining the exposure by means of the meter, the reading should be made from the operative field itself and not from the surrounding sheets. After the incision it may be well to increase the diaphragm opening, as the color change from flesh tints to deep reds and yellows occurs.

If the line of vision is unavoidably blocked, even for a short time, by assistants or nurses, these portions should be cut from the film.

Any amateur at all conversant with indoor photography should make successful pictures in the operating room, as the illumination there is sufficient for most purposes.

Animation.—A much more fascinating field and one seldom cultivated by amateurs is animation, which affords an excellent means of demonstrating the action of instruments, apparatus, anatomic structures and mechanical processes with a facility much greater than is afforded by blackboard demonstrations or lantern slides. "Animation" is the term applied to the cinephotographing of inanimate objects which are moved between exposures or "frames" so as to give the appearance of spontaneous movement.

The complicated animation seen in the professional black and white comedies entails endless thousands of drawings, but this is not the case with the type of animation here referred to. Much can be done with cutouts made from drawings or photographs

which are laid upon a horizontal surface and photographed from above, and which are moved between exposures made one frame at a time. For this work a firm vertical support is necessary. Few tripods tilt to a vertical position, but an excellent stand can be made from an ordinary steel kitchen chair with a back rest. The wooden seat is removed from the metal ring and the camera is screwed to the back of the chair, through which a hole has been drilled. The chair with the camera attached is then set on the working table. Before photography is begun, the fields of the various lenses should be mapped out on the table and the fields of the finders designated by dotted lines to permit centering diagrams. Two or three thicknesses of gauze attached to the legs of the chair serve as diffusing screens and prevent harsh shadows. Two ordinary 200-watt lamps, of the type used on title-writers, set on the table, one on each side of the chair, supply sufficient illumination, or the 500-watt lamp with reflector may be set very close to the field to be photographed. Two cheap mirrors, a foot square, set against the sides of the chair, conserve much light and prevent cast shadows at the edges of the cutouts which destroy the illusion of flat drawing. A remote shutter control is convenient and reduces vibration appreciably, but is not indispensable. A mechanical type of control is preferable to a pneumatic bulb control, as the latter is often uncertain in its action. The cutouts are made from drawings executed in wide lines with lusterless black ink on a dull white surface. They should be cut from stock sufficiently heavy to prevent curling from the heat of the lamp. Three-ply cold-pressed Strathmore board is suita-

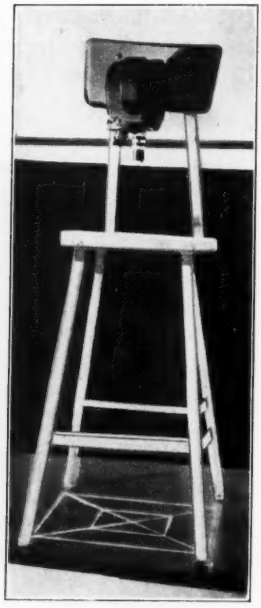

Fig. 1. A convenient camera stand for reproduction and animation.

ble. They should be laid in position in the lens field as indicated by the lines on the table and focused with the utmost care. The exposure also must be carefully estimated, as slight over-exposure

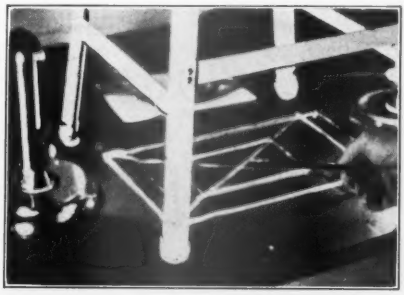

Fig. 2.* The lens fields are mapped out on the table to permit of easy centering.

results in a flare and detail is lost, while slight underexposure grays out the background and results in a muddy picture.

With the cutouts laid in the starting position, it should be determined how much distance each moving part is to cover in one

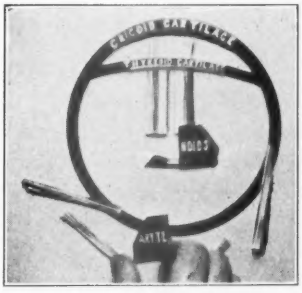

Fig. 3. Cutouts for animation.

second of time. With a normal exposure of sixteen frames a second, it will be necessary to execute sixteen individual movements of the cutout to cover this distance. If the distance is to be covered in one-half second, eight moves are made, and so

*Figs. 2, 3 and 4 are enlargements from 16 mm. film.

on. With the cutouts in the starting position, the camera shutter is tripped so as to permit the exposure of a single frame. The cutouts are now moved and another single frame is exposed. After another move comes a third exposure, and so on until the entire motion has been executed. It is well to use movable arrow cutouts freely to accentuate the portions of the diagram which are of especial interest and to attract the eye to these points.

The gravity catch which holds down the exposure button in some makes of camera has an exasperating way of interfering with individual exposures. It is strongly recommended that this gadget be taken out of the camera by someone conversant with this operation, otherwise the motion may be jerky and the illumination uneven. One type of camera has a very efficient stop for making pictures one frame at a time.

Diagrams may be clarified by drawing them piecemeal as the film is made. This can be done either by photographing the hand as it wields the pen or by stopping the camera while additions are made. In this way a dotted line may be made to project itself across a drawing; important parts of a mechanical apparatus may be sketched in after the rest is drawn, to call attention to them; the pen may be shown actively labeling details. If such additions require a long time to letter or sketch, the camera should be stopped at intervals, while the sketch progresses, to avoid monotony. Or the film may be run eight frames to the second (half speed) through the camera and projected at ordinary speed, which will give the effect of very rapid drawing or lettering.

In all "stop-motion" work it is well to remember two things: First, that the total time of a given movement should be worked out before exposure begins. It requires a relatively long time to complete sixteen movements and sixteen exposures, and unless the action is previously planned, it is difficult to realize that only one second of film has gone by; hence the first experiments are apt to prove too rapid and too fleeting. Second, owing to the inertia of the moving parts of the camera, the first frame of a sequence is always overexposed. As all the frames in stop-motion work are first frames, allowance for this overexposure must be made by using the next smaller stop of the diaphragm. If, in the midst

of stop-motion work, a strip is allowed to run at ordinary speed, the iris should be temporarily opened accordingly. Stop-motion work lends itself particularly to the demonstration of instruments or other mechanical apparatus which may be moved a little at a time between exposures, and it is sometimes advisable to combine an instrument with a black and white cutout animation.

Diagrams, animated or otherwise, can be applied to living subjects to demonstrate the outlines of organs or anatomic structures, by punching out dots from gummed paper (or cutting dashes from it) and applying them to the skin. Line drawings on the skin with a deep brown or black dermatograph pencil photograph well.

Roentgenograms.—Up to the present time no method has been devised for making true roentgenographic motion pictures. The illumination of the fluoroscope is far below that required for

Fig. 4. Applying a dotted line to a living subject.

producing an image on the film. The illusion of motion has sometimes been produced by making a series of ordinary roentgenographic films at intervals and photographing these by transmitted light upon successive frames. In this way, for example, the growth of a skull has been recorded over a period of years and reproduced upon a strip of film which projects the whole story in a few seconds. It is often useful, however, to photograph a single roentgenogram in order to enlarge it sufficiently to permit of superimposing diagrams or otherwise accentuating it; or merely as a still, incorporated in a demonstration. The most

satisfactory way of making such photographs is to set the camera before a viewing box and to illuminate the film by transmitted light. Attention may be centered upon a particular area, such as a sinus, by mounting an opaque card of the desired shape upon the glass behind the film. This will appear in the movie as a solid black area. In like manner, arrows may be cut out and mounted behind the film.

Photostats.—Negative photostat prints of diagrams in which the background is black and the line drawing is white, lend themselves particularly well to cinematography. They are sharp and clear and produce less eye strain than do the highly illuminated white backgrounds.

Cinemicrography.—Cinemicrography is a highly specialized art and requires expensive apparatus. The advantage of recording microscopic movement, and moreover, of being able to repeat it at will and of stopping the motion at any desired point is so great that the cost may not be prohibitive.

The chief problems in micrography are illumination and focusing. The preferable illuminant is the arc lamp, as it is often difficult to obtain sufficient illumination from incandescent lamps. Here again the new superspeed film is overcoming what were almost insurmountable difficulties. The chief annoyance with the arc is its tendency to shift its position in the crater of the carbon and thus to throw the point of maximum illumination to one side or even entirely out of the field. To overcome this, it has been suggested that an aplanatic system of lenses be introduced between the arc and the mirror of the microscope, which will permit the arc to wander about, without varying the ultimate illuminating beam. Dark field illumination may be necessary. Apochromatic objectives should be used in the microscope.

In order to insure exact focusing at all times, even while the exposure is progressing, it is necessary to introduce some device between the microscope and the camera by which a small percent of the beam can be split off for observation. R. F. Mitchell has suggested a highly efficient system, which contains two such units. In his arrangement a Goerz sharp-focusing device is introduced into the lens opening of the camera and a microphot over the ocular tube of the microscope. The photographic and the

microscopic halves of the outfit are connected through these two accessories. The advantage of this arrangement lies in the fact that the mirror of the direct focusing device can be entirely withdrawn from the system after sharp focusing is done, thus eliminating any loss of illumination at that point. The microphot, which splits off only about 5 per cent of the illuminating beam, can be used for observation during the exposure. It goes without saying that the focuses of the direct focuser and the microphot must be accurately matched at the beginning of the experiment.

When stop-motion work is required with this apparatus, another unit is added to make timed electrical contacts by clockwork, which operate the camera shutter one frame at a time.

A motor-driven camera has been developed, upon a rigid base, which is adaptable to the microscope and which can be regulated to expose the film at measured intervals, ranging from sixteen to the second to one every four minutes.

"Stills."—Frames from 16 mm. film can easily be enlarged as still photographs to as much as three by four inches, if the negative is sharp. Stills made in this fashion have several advantages. To begin with, the action can be caught at the precise moment required. Second, such a photograph represents a phase of an actual movement and not a simulated one, as is the case when the subject is posed immovably in some position representing action. Third, a series of such stills may be made representing successive stages in the progress of a movement which, combined, will outline the complete movement better than any single photograph can do. It often assists matters to mount them in a vertical strip in imitation of a strip of film, which at once suggests progressive motion to the student. Such strips are especially useful as illustrations for technical publications.

One might go on indefinitely; but the foregoing will suggest to the amateur cinematographer that his art is limited only by his ingenuity. It is but fair to warn him that his first reel is more than likely to inoculate him with an incurable disease.

1010 Beaumont Bldg.

The Scientific Papers of the American Bronchoscopic Society

SYMPOSIUM ON CANCER OF THE ESOPHAGUS: DIAGNOSIS AND TREATMENT.

LXXI.

I. ROENTGENOLOGICAL ASPECTS.*

(By Invitation.)

Edwin C. Ernst, M. D.,

St. Louis.

Esophageal cancer is indeed a serious problem when from 6 to 10 per cent of the reported fatal malignancies occurred in this region. Perhaps even more important is the fact that so few five-year curves have been authentically reported in the literature of today.

Since the most frequent complication of this disease is the involvement of the respiratory system, either by direct extension toward the lung; by perforation into the trachea and bronchi, with or without hemorrhage, general exhaustion and starvation phenomena, or by the more rapid bronchial pneumonic infiltrations, but rarely encounter early metastasis into distant organs, and it would seem as though we were dealing with a disease that should be more hopeful. This disease, in the early stages at least, presents only local manifestations and therefore, logically, we should expect a greater number of permanent cures.

Perhaps we see these patients only in the very late stages; or, more probably, the disease with which we are dealing is indeed a critically malignant one, and the surrounding structures are infiltrated almost from the very beginning in a manner or by a method not in keeping with our present understanding and knowledge of the physiologic and pathologic phenomena involved.

*Read as part of a symposium at the meeting of the American Bronchoscopic Society, St. Louis, June, 1931.

On the other hand, occasionally these esophageal lesions in reality are very radiosensitive. This fact should stimulate us to reflect and review our past successes and failures and not become too despondent. First of all, let me emphasize this one important phase of the problem.

My experience with the diagnostic and therapeutic problems of this disease has led me to appreciate the fact that perfect "team work" between the surgical, medical and radiological consultants is of primary importance for the future developments toward a more successful handling of this problem, and the privilege accorded me of appearing before you is proof of the fact that you likewise realize the advantages of co-operation and the liberal exchange of ideas and experiences.

This is not only advantageous from an early diagnostic standpoint, but is equally applicable to the selection of the proper and most beneficial palliative measures in the treatment of even the more advanced stages of this disease.

In view of the foregoing premises relative to the problem of esophageal cancer, I wish to emphasize at this time two very essential and important phases of the future possibilities of probably improving our present efforts in combating this disease.

First of all, every one of us should continue to work in the interest of more thorough and complete examinations, employing all of the available new methods or technical procedures for stimulating the possibilities of earlier diagnosis, and secondly, from the standpoint of treatment, to likewise stimulate your interest in the several possible palliative measures at our disposal in the treatment of this disease.

I realize that even under the most favorable and ideal conditions there are many reasons for our past failures in the early diagnosis and treatment of this disease.

Esophageal cancer is unfortunately rarely detected early. Perhaps the lack of ability and technical skill of the bronchoscopist and radiologist in the past have shouldered at least a portion of this responsibility.

Inaccessibility of the average esophageal cancer lesion, both from a surgical, as well as a radiation therapy standpoint, has been one of several contributing factors to our past failures.

The many apparent complicated methods of extension, unfortunately adopted by the original esophageal cancer lesion in a region so close to the vital respiratory organs, has limited the necessary amount of radium that could possibly be safely employed as well as handicapped the surgical approach for a complete removal of the lesion.

Many of our failures may also be attributed to the fact that a relatively small intrinsic cancer lesion is so frequently surrounded by a much larger extra-esophageal mass.

These esophageal lesions are nearly always infected tumors from the very beginning, and in consequence may involve the glandular structures early, as secondary inflammatory reaction, and in sequence these infected glands easily become the seat of cancer foci.

However, we should not become too discouraged. In the past esophagoscopy studies have been of the greatest value.

In the future the skill of the esophagoscopist may help to eliminate some of these reasons for our failures and indirectly improve our end results by the earlier recognition of these infiltrations within the esophageal lumen.

At times the X-ray examination in the extreme upper esophageal area is not very satisfactory from either a positive or negative roentgenologic standpoint, due to irritation phenomena or spasms, and therefore too much dependence must not be placed upon any negative X-ray finding of a suspicious early lesion.

Esophagoscopic studies may prove of greater value in such cases.

In the midesophageal region of the thorax many benign glandular enlargements, either aneurysmal, simple inflammatory, tubercular, or specific infections should, first of all, be excluded, fluoroscopically as well as radiographically, by means of lateral and semilateral X-ray films.

Occasionally, extreme scoliosis or kyphosis of the thoracic spinal vertebræ show an abnormal esophageal position or pressure defects, but it is especially important to remember the possibility of a localized thoracic abscess arising from a tubercular extension of the spine.

Unless a very complete roentgenologic study is made, this latter possibility of a chronic spinal lesion may be easily overlooked.

Unquestionably, we all realize that the earlier a case of esophageal cancer is discovered, the closer do we approach a cure and the more effective will be the employed palliative measures.

Consequently we must continue to redouble our combined efforts in the direction of a more advantageous and correct interpretation of the very earliest signs, symptoms or suspicious history suggestions of esophageal cancer. This should remain the keynote of our endeavors. Equally important to us, as radiologists and esophagoscopists, is that we must be equal to the occasion and prove ourselves to be sufficiently alert and equipped to correctly interpret such suspicious clinical observations with a reasonable degree of accuracy, when we are called upon for help.

By means of the illustrations, Figs. 1 and 2, the lung and mediastinal structures may be examined not only fluoroscopically but also radiographically, in every conceivable position, namely, the anteroposterior, semi-oblique, direct lateral, vertical and the horizontal positions, with every protection to the radiologist. The very moment that a defect is noted or even suspected, a film is made of the esophageal lumen exactly as visualized fluoroscopically.

Every routine examination must include an intensive and careful fluoroscopic study of the act of swallowing, during the passage of the opaque media along the lumen of the esophagus with various types of opaque media. The following standard opaque emulsion has been most helpful to us in our work. Barium sulphate is added to meet the needs of condition or lesion requiring interpretation:

Barium sulphate	400, 0
Tragacanthæ	3, 0-5, 0
Amylium acet.	gtt. v iii
Spir. 90%	10.0
Aqua	800.0

The size, shape, outline and position of the esophageal lumen, the deglutition and peristaltic phenomena, the degree of obstruction, if present, are the primary observations to be recorded.

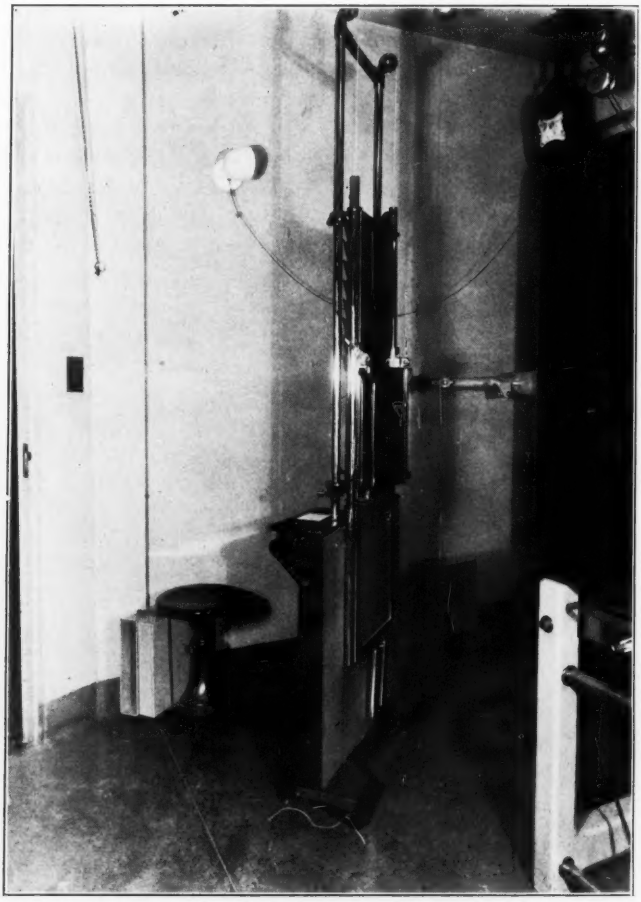

Fig. 1. Protectoscope or movable fluoroscopic screen devised for the vertical esophageal studies and instantaneous radiographic films affording complete protection to the radiologist.

Fig. 2. Showing the patient in position. The entire esophageal tract can be visualized and any portion may be radiographed instantly by inserting a film in the cassette holder and pressing a switch so that the amount of X-ray current is increased ten fold.

The absence or presence of small marginal defects or flecks of barium adhering to the esophageal walls, all such observations must be checked and double checked in the various positions above described.

Should the esophageal lumen indicate obstruction, then the region immediately above and below this probable lesion or narrowing will require careful analyzation and interpretation.

The patient is then placed in the "Trendelenberg" position to facilitate the filling of the lower limits of the partially obstructed esophageal lumen so that we may likewise identify the upper margins of the distal portion of the normal esophagus, and thus be in a position to accurately estimate the degree or extent of the tumor involvement.

We are especially partial to the "horizontal" position for the added help afforded in visualizing early esophageal lesions.

No roentgenologic esophageal examination is complete without taking advantage of this procedure.

In the study of the upper esophageal region, the possibility of tracheal or laryngeal new growths, inflammatory tumors or goiter must be given consideration.

Whenever a diverticulum of the esophagus is suspected, it is always a safe procedure to, first of all, have an X-ray study made prior to any instrumentation of these unknown regions.

Instantaneous serial films in the various directions may then give additional information as to the type and character of the abnormal pouching, and roentgenologically it may be possible to determine whether or not this type of diverticulum is due to pressure from within or retraction phenomena from without.

Frequently such a diverticulum is accompanied by extrinsic esophageal adhesions and retraction of the pouch, or may be due to pressure from within the lumen.

In the study of diverticuli, it is always important to employ sufficient opaque paste so that the "pouching" may show the usual overflowing characteristics into the normal esophageal lumen.

On several occasions I have been asked to corroborate the supposed findings of a complete esophageal obstruction, but fortunately only discovered an enlarged diverticulum incompletely filled.

The internists who made these examinations employed too small a quantity of bismuth, merely filling the diverticulum, and thus the diagnostician reached his final conclusion too early.

Of course, we must at all times give due consideration to local esophageal irritation phenomena due to impacted foreign bodies, localized ulcerations, simple tumors, polyps, tubercular and syphilitic infiltrations, and from a roentgenologic standpoint the final differential diagnosis may be difficult.

Foreign bodies or hemorrhagic blood clots may prove very disconcerting at times, even though the history of the esophageal obstruction is atypical of cancer.

Fig. 3 is typical of an obstruction following a "gin party" in which the symptoms persisted over a period of several days. Repeated roentgen findings showed no apparent changes in the degree of obstruction. An esophagoscopic study by Dr. A. McMahan revealed a supposed blood clot, but the causative factors could not be determined. Several days later the patient obtained relief after coughing, and Fig. 4 again shows a normal esophageal lumen.

We must, therefore, not be satisfied with the roentgenologic procedure, but pray for additional help from the bronchoscopists.

The question of excluding cardiospasm is indeed important. Rosenheim, in 1895, by means of the esophagoscope, and Rumpel, in 1897, by means of the fluoroscope, were the earliest workers to describe this idiopathic dilatation of the esophagus.

Since then cardiospasm has been studied by many observers, and it would take the entire morning to thoroughly cover this phase of the subject.

However, the differential diagnosis is reasonably safe and accurate.

The characteristic typical dilatation phenomenon of the esophagus above the spasm is characterized by an oval appearance and relatively smooth shaped round corners, as well as consideration given to the location of the constriction. All of these observations are important factors of value in the differential study of esophageal cancer.

It is nevertheless true that even a careful observer may be misled by these signs.

Fig. 3. Partial but continuous obstruction due to blood clot.

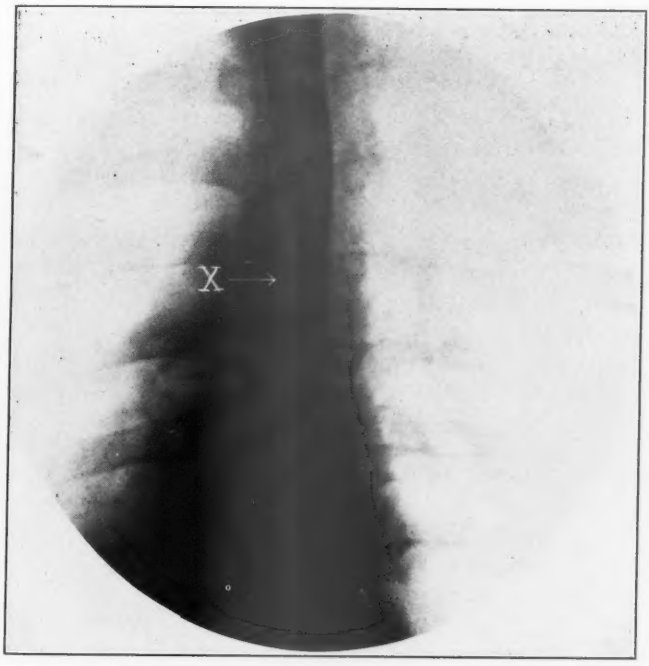

Fig. 4. Several days after esophageal studies by means of the esophagoscope this blood clot passed without showing any visible wall defect.

In our work we are very cautious and recommend a rechecking of our original observations, after the administration of an antispasmodic, since we realize the importance of this necessary added corroborative evidence before venturing a final diagnosis.

The treatment of cardiospasm by forced dilatation is a heroic procedure, and we are especially anxious to, first of all, reasonably exclude the possibility of any other lesion.

The question of treatment of this condition will always remain more or less an individual problem.

In the past the preferred method of therapy has been largely limited to measures, namely, gastrostomy, dilatation of the esophageal stricture, with or without radium intubation, and last, but not the least important, the employment of external radiation, by means of carefully measured high voltage X-ray applications.

Occasionally, but rarely, surgical removal is given consideration.

From the standpoint of gastrostomy, there are advantages and disadvantages to advising this preliminary procedure prior to administering treatment in the early cases.

In other words, we are not enthusiastic about the added help of this procedure except in special cases or where there is complete obstruction.

The question of dilating the local stricture and applying, by means of the various intubation methods, radium radiation; all such measures depend largely upon the individual problems in a given case, taking into consideration the histologic type, extent and character of the local cancer involvement, etc.

I personally must admit that I have been employing these direct radium applications with ever-increasing infrequency.

Unless the lumen is sufficiently patent to allow the introduction of a moderately large well filtered tube of radium with a maximum amount of rubber, without producing much trauma, I am reluctant to advise these measures based on my past experience and results, as shown in the radiogram, Fig. 5, also Fig. 6, this procedure is questioned. The latter patient has responded beautifully to external radiation therapy for several years. Unquestionably there is a large mass still present in the mediastinum surrounding the intra-esophageal lesion. In Fig. 7, on the other

Fig. 5. Extensive esophageal cancer.

Fig. 6. Moderately advanced esophageal cancer lesion. Deep X-ray therapy alone have shown marked palliative results.

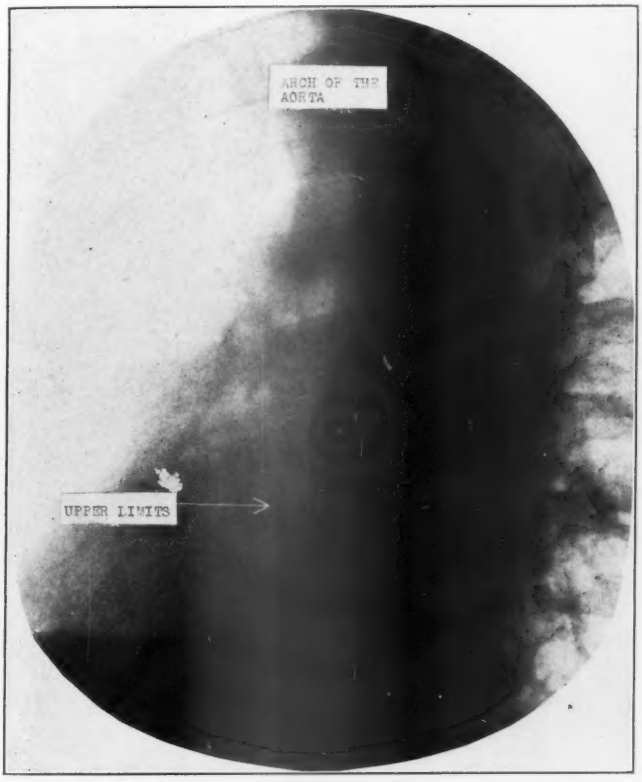

Fig. 7. Early esophageal cancer lesion. Combined methods of local highly filtered radium and deep X-ray therapy have relieved all symptoms.

hand, the early cancer involvement has been benefited by the additional radium application.

When radium is employed, we prefer the highly filtered radium (one or two millimeters of platinum) applied every few days for a relatively long period of time. The total dosage varies with the size of the radium applicator as well as the filtration.

Needless for me to state, it is also desirable that the full amount of radium, either 12½ or 25 milligrams, in tandem, should radiate the upper and lower limits of the lesion so that the probable extra-esophageal tumor mass may receive the benefit of cross-firing gamma rays.

However, if the area is greater or longer than 5 cm., or the lumen of the lesion is extremely narrow and tortuous, I feel that the value of highly filtered radium applicators are overrated. Unfiltered radium is always contra-indicated. Even gold filtered radon seeds are difficult to apply except in the few selected groups of cases.

In all of these cases, however, deep X-ray therapy precedes the application of radium to the local lesion.

Contrary to the occasionally expressed unfavorable opinion regarding the value of deep X-ray therapy, we find this procedure to be not only of value but almost indispensable.

The average moderately filtered, mildly penetrating X-ray beam could not hope to direct sufficient energy into the cancer area to be effective from a palliative standpoint.

On the other hand, it is equally true that the ideal intensive type of deep X-ray radiation occasionally does have little or no effect upon a few of the more resistant types of tumors in the advanced stages of the disease. This is frequently due to the presence of other complications or distant metastasis.

The local esophageal constriction, roentgenographically, may present very few changes over a long period of time, nevertheless the general physical condition of the patient, as well as his moral make-up, show marked improvement after X-ray treatment alone. The average esophageal lesion should receive no less than 900 "r" units with back scattering through four ports of entry. We routinely employ 200 K. V., at 60 cm. distance, with a filtra-

tion of from .5 to 2 mm. of copper, depending upon the size and density of the thorax.

The question of reducing the pain and oppressive symptoms is quite another problem.

In about half of our cases these latter symptoms show worthwhile improvements.

In the routine or average esophageal cancer we advise at least the employment of deep X-ray therapy, no matter what treatment may be instituted at a later date.

Especially is this desirable prior to the application of radium or radon seeds when directly introduced by means of the esophagoscope.

Someone recently stated that the important contraindications to X-ray therapy was the possibility of lung injury.

Fortunately, I have never observed any such changes, but in doing so I wish to emphasize that both the unfiltered or semifiltered application of radium or X-rays are distinctly contraindicated as therapeutic measures in all types of esophageal cancer. Under such conditions the skin and lung structures receive the greater quantity of the radiation, while the tumor receives correspondingly less.

However, if the actual X-ray beam is carefully measured, and directed toward the tumor, preferably calibrated on the absorption bases in "r" units, and in addition every effort made mathematically to correctly direct the crossfiring of the respective beams so that the maximum absorption of these rays may be made possible, and the X-ray treatment is continued over a period of from three to six weeks (not administered by means of a few large doses), and furthermore that such radiations are directed through at least three or four portals of entry in the direction of the tumor. then and only under these conditions will the worth-while improvements in a large series of cases of inoperable cancer become apparent.

BEAUMONT MEDICAL BUILDING.

LXXII.

II. ESOPHAGOSCOPIC ASPECTS.*

CHEVALIER JACKSON, M. D.,

PHILADELPHIA.

Cancer as a word has come to mean any form of malignant tumor. As applied to the esophagus malignant growths are most often carcinomatous, rarely sarcomatous, and still more rarely endotheliomatous. The differentiation among them can be made only by histologic examination of a specimen of tissue. The general diagnosis of all three varieties is the same.

DIAGNOSIS OF MALIGNANT NEW GROWTHS OF THE ESOPHAGUS.

The diagnostic features usually mentioned are a history of progressively increasing difficulty in swallowing, weight loss, pain, emaciation and cachexia. These are suggestive of malignant disease but they are not diagnostic, and moreover when really associated with cancer are hopelessly late. In some cases they are directly misleading.

The esophageal bougie as a diagnostic means is always negative early, inconclusive later, inferential at best, and often fatal.[1]

A diagnosis by the classical symptoms of pain, emaciation and cachexia is hopelessly late, and even these symptoms may be present in the absence of cancer. Pain, a prominent symptom of cancer elsewhere, is always late in cancer of the thoracic esophagus and may be totally absent to the end.

There are only two important diagnostic means, namely—

1. Roentgen ray examination.
2. Diagnostic esophagoscopy, with or without biopsy.

The roentgenologic diagnosis will be presented by my distinguished colleague, Dr. Edwin C. Ernst; but I deem it my duty to say that we have found the roentgenologist's report, when positive, correct in over 90 per cent of the cases. Negative reports have been correct in about 85 per cent of the cases. In all

*Read as part of a symposium at the meeting of the American Bronchoscopic Society, St. Louis, June, 1931.

of the patients coming to the Bronchoscopic Clinic complaining of any abnormality in swallowing, roentgen ray examination precedes endoscopy; and, it might be parenthetically added, the same applies to all other patients.

Esophagoscopic Diagnosis.—By esophagoscopy, endo-esophageal cancer can be diagnosticated not only early but with the absolute certainty essential in order to get the consent of a comparatively well man to an operation he may not survive. Endo-esophageal cancer can be diagnosticated just as early, just as quickly and just as certainly as cancer of the cervix when an opportunity for esophagoscopy is afforded early.[3]

From the viewpoint of the esophagoscopist, there are two classes of cases: (1) peri-esophageal growths, and (2) those that are endo-esophageal by origin or extension. The endo-esophageal cases preponderate to the extent of nearly 90 per cent in the records of the Bronchoscopic Clinic.

Esophagoscopic Appearances.—The endoscopic image in cancer of the esophagus will depend somewhat upon, (a) the stage of the growth at the time of examination, and (b) the location of the growth, endo- or peri-esophageal. A very early endo-esophageal lesion may be in the form of one or more nodules covered by eroded epithelium; later the nodule ulcerates and fungations appear. A fungating, bleeding ulcer is the type of lesion must frequently seen in the esophagoscope. The fungations may be covered with a very thin layer of epithelium, but this does not give the appearance of mucous membrane; sometimes they are whitish or reddish, papillomatous looking masses, smeared with blood which fills the interstices.[7]

In the peri-esophageal growths that have not yet intruded, the sudden change from the beautiful, soft, velvety, pink mucosal folds that slip away from in front of the tube mouth to the firm infiltration of a malignant process is so characteristic to the eye and touch of the esophagoscopist as to be almost unmistakable. In some of these cases blood will be seen oozing up from below. The most important point is the education of the eye and touch to the appearances and feel of the normal. Form more often than color is the criterion of the malignant image. Only experience can differentiate between the firm resistance of the as yet un-

relaxed cricopharyngeus and the infiltration of cancer of the hypopharynx or cervical esophagus. In the thoracic esophagus there should be less difficulty on the part of anyone familiar with the appearances of cancer of mucosal surfaces.[2]

Differentiation from peptic ulcer can be made only by esophagoscopic biopsy; and the same may be said of most other lesions.[5]

Esophagoscopic Biopsy.—A specimen can very easily and safely be taken in all cases in which there is present a nodular fungating or ulcerative lesion. Anything that will come away readily with cupped forceps is not normal. Very sharp cutting forceps may be used, but if as sharp as they should be the sense of touch is not so good a means of diagnosis. In cases of infiltrative growths covered by normal mucosa, such as the peri-esophageal malignant lesions, it is better to wait a month or two for intrusion, rather than to bite through normal esophageal wall to reach a tumor primary in the mediastinum.

PROPHYLAXIS.

Prevention of cancer in the present state of our knowledge consists in avoidance of irritation. As applied to the esophagus this is to be done by the early diagnosis and cure of all curable disease of the esophagus, plus the avoidance of irritating and excessively hot foods and drinks. Hot tea and coffee are probably the most commonly used of these irritants. Many of the cancers encountered at the lower end of the esophagus, in the esophagoscopic examinations in the Bronchoscopic Clinic, were the upper edge of extensions from malignant growths in the stomach; this clinical fact suggests that the prevention of cancer of the esophagus is closely allied to prevention of gastric cancer. In a few cases there was good reason to believe that cancer of the esophagus was secondary to peptic ulcer of the esophagus, and in quite a number cancer appeared at the edge of scars left by healed ulcers. The sequence of cancer following ulcer is not as common in the esophagus as it is in the stomach. Prophylaxis of mortality from esophageal cancer is considered apart from incidence, here as elsewhere, and rests fundamentally on early diagnosis. This can be made easily and positively by esophagoscopy preceded by roentgen ray examination, and in no other way. So long as the patient is

allowed to drift on to certain death under erroneous inferential diagnosis of "spasm" and "globus hystericus," just so long will it be useless to try to lessen the mortality of cancer of the esophagus.

CURATIVE TREATMENT.

Until some now unknown method of treatment of cancer of the esophagus shall have been discovered we must look to the surgeon for hope of cure.[3] Nor should the gravity of the procedure deter us, since we know that the disease involves 100 per cent mortality. There is good reason to believe endo-esophageal cancer is curable, surgically, at some levels at least, if a sufficiently early diagnosis is made. It is of a relatively mild degree of malignancy and it metastasizes slowly and very late in the progress of the disease. The fundamental reason for the thousands of agonizing deaths from cancer of the esophagus is false education.[4] The mind of the medical student and the medical profession is saturated with three erroneous ideas: (1) that esophageal obstruction is often spasmodic; (2) that cancer of the esophagus is to be detected by obstruction to the passage of a bougie; (3) that the diagnosis of cancer is to be made by the history of the case and the symptoms of dysphagia, pain, cachexia and emaciation. These are the reasons why the surgeon never gets a chance to cure esophageal cancer while it is still a local process. When every patient mentioning the slightest abnormal sensation in the cervical, retrosternal or epigastric region is considered probably not neurotic but possibly cancerous, surgery will show better results. When every such patient shall be referred at once to the roentgenologist for fluoroscopic and roentgenologic study of the esophagus, esophageal cancer will be discovered early and a new era in esophageal surgery will have been established. The surgeon should be encouraged in his efforts to perfect the technic of esophagectomy and also in that of the external implantation of radons in cancer of the esophagus, cervical, thoracic and abdominal. Of course the full gravity of any external surgical operation must be explained to the patient and his consent obtained. Radons tethered with silk threads may be inserted esophagoscopically with great accuracy; but so far the method has not been demonstrated to be more than palliative.[8]

PALLIATIVE TREATMENT.

The most important thing in palliative treatment is to begin it early. An early diagnosis is just as essential for successful palliation as for attempts at cure. This is often overlooked. The patient should not be allowed to live in a fool's paradise but should be informed of the nature of the illness, not only that he may have ample time to arrange his affairs, but also that by his co-operation in early palliative measures his life may be prolonged by many months, even years.

Medical Care and Management, Including Diet.—It is of fundamental importance in prolonging life and palliating discomfort that dietary and medical management be instituted early; for this an early diagnosis is essential. It is much easier to retain than to regain lost weight and vitality; moreover, in mismanaged diet there always lurks the danger of acidosis, depression, melancholy or loss of morale: conditions which result in irretrievable loss of vitality. Fatigue and worry should be avoided, plenty of sleep insisted upon. Elimination by bowels, skin and kidneys should be gently but continuously increased, and, above all else, a careful supervision of the diet should be maintained. Unless guarded against, in nearly all patients, as swallowing becomes difficult, two errors will occur: (1) improper or improperly prepared food will lodge, causing esophagitis and swelling that increase the obstruction; (2) the patient will drift into a steady diet of meat broths, milk and raw eggs. An abundance of vegetables and raw fruits are essential; but, of course, they must be sieved or strained so as not to lodge. The mesh of the sieve or strainer should be about 25 to the inch. Any food that can be pressed through a wire mesh of this size will go through a very small fistulous lumen or the smallest tube. Milk should not be given at or near the same time as curd-producing foods, because curds will obstruct a small lumen.[6]

Dilatation.—In order that the dietary essentials may be used effectively it is necessary to maintain a lumen through the cancer. This can be accomplished by gentle, careful dilatation with the esophagoscope, under guidance of the eye, using the steel-stemmed esophagoscopic dilators. A small filiform should first

be passed to make sure there is a lumen to be followed. Then increasing sizes are used up to the desirable degree of dilatation. Repeated seances and accurate placing of the dilators in the lumen render this esophagoscopic dilatation quite safe in trained hands. We have never had a fatal result attributable to this method. More rapid results are obtainable by using large olive-shaped metallic dilators threaded over a string, but rupture of the esophagus or direct perforation is inevitable in a certain small proportion of the cases.

Intubation has given good results in some cases. Our soft rubber tube has some advantages; but good results have been obtained with silk woven and metallic tubes. The choice of tube should lie with the operator in the particular case. Intubation should not be attempted until the presence and location of the fistulous lumen has been determined by esophagoscopy.[6 8]

Gastrostomy.—An efficient and relatively safe palliative measure is very early gastrostomy to stop the mechanical irritation of passing and lodging food, and the chemical irritation of fermenting and stagnant food and secretions saturated with oral infections. If an early gastrostomy be done and the esophagus be put at rest with only water by mouth, saliva will go through for a long time. If the patient be allowed to go far in food and water starvation he cannot be brought back, even after gastrostomy, and the mortality of the operation will be high. Intubation will postpone the necessity for gastrostomy but will rarely obviate it, unless the patient is to be allowed to die of hunger and thirst. As with all other palliative measures in these cases, gastrostomy is almost always done too late, consequently general statistics of the operation are very misleading, both as to operative mortality and subsequent duration of life. Acidosis and dehydration are the two chief causes of operative mortality. These are due to postponement of operation. When dehydration is present, preparatory treatment with large quantities of water, very slowly given, preferably by colonic drip, will greatly diminish operative mortality. A little sodium bicarbonate may be added to the water, but albumen and other foods are worse than useless because they cause intolerance with consequent expulsion of the all important water. The decision as to whether or not a gastrostomy is to be done in cases of cancer of the esophagus should be left

to the patient in the particular case after the situation has been explained to him. If he prefer to follow the advice of the physician the latter may state what he thinks his personal choice would be under similar circumstances. Individuals who set great store by the pleasures of the table would rather prolong at all hazards the swallowing of food; others who have a life's work to finish and consider time at table largely wasted would prefer utmost prolongation of life, regardless of the form of food or manner of administration.[3]

Deep Roentgen Ray Therapy.—Of the palliative measures directed against the growth itself, deep Roentgen ray treatment has yielded the best results in our experience; but only when used early. One of the saddest mistakes is to radiate a patient in an advanced state of emaciation and cachexia. He will become toxic and usually will last but a few weeks. In marked contrast to this, we have seen a small early esophageal malignant growth arrested for many months, occasionally a year or two, in a few instances for as long as five years, by deep Roentgen ray therapy combined with the dietary and other palliative measures mentioned above. Here again we come back to the fundamental necessity for early diagnosis. It should be repeated for emphasis that this diagnosis can be obtained only by esophagoscopy and roentgen ray study in every patient complaining of the slightest abnormality in swallowing. Furthermore, the diagnosis of a "spasm" should never be made until the esophagoscope has ruled out an organic lesion.

Radons may be inserted with accuracy by means of the esophagoscope. They can be left indefinitely in the tissues and usually disappear from esophageal cancers, probably by the gastrointestinal route; but if deemed better to remove them after they are spent, this can be readily accomplished by attaching a short black silk thread to the radon, which should have an eye for the purpose. These threads are very readily found with the esophagoscope and the spent radons may thus be removed with the side-curved forceps. The radons should be placed at slightly different distances from the mouth to avoid overlapping of the radiation. The results to be hoped for in therapy with radons is a temporary arrest of progress of the growth and an increase of lumen.

CONCLUSIONS.

1. Most patients with cancer of the esophagus are treated under an erroneous diagnosis of "spasm" until the growth is far advanced.

2. Esophagoscopy is the only means of making with certainty a very early diagnosis of cancer of the esophagus.

3. Roentgen ray study is a most valuable means of diagnosis of malignant disease of the esophagus.

4. Neither esophagoscopy nor roentgen ray study should be omitted in any case of the slightest abnormality in swallowing: both are essential for diagnosis.

5. A diagnosis of spasm of the esophagus should never be regarded as more than tentative until an organic lesion has been ruled out by esophagoscopy.

6. The operation of esophagectomy has never had a fair trial because the esophagoscopist and the roentgenologist very rarely have an opportunity to make an early diagnosis. The surgeon should be encouraged in his efforts to perfect the technic of operation and also that of external implantation of radons in cancer of the esophagus, cervical, thoracic and abdominal.

7. Deep roentgen ray therapy is the most efficient of the palliative measures directed against the growth itself; but only when instituted early and combined with proper medical care and management, especially dietary management. It is a mistake to radiate a patient far advanced in emaciation and cachexia. Increase of toxemia will shorten his days.

8. Radium and radons in our hands have been, on the whole, less effective than deep roentgen ray therapy; but their use is advisable in some cases.

9. Earliness in commencing palliative treatment is quite as essential as in any attempt at cure. The difference in prolongation of life between palliative measures begun very early and those instituted late usually amounts to many months, sometimes years.

10. Gastrostomy, intubation and esophagoscopic dilatation all have advantages and disadvantages, but each and all require early performance to yield satisfactory results. This requires early diagnosis, which is rarely done, for lack of early esophagoscopy.

11. The choice as among gastrostomy, intubation and dilatation should be left to the patient after the situation is explained to him. He who sets great store by the pleasures of the table will prefer to have swallowing maintained to the last at all hazards. Those who have a life's work to finish and consider time at table wasted will prefer utmost prolongation of life with least possible risk in the means employed.

12. One of the most essential elements in palliation is the very early institution of medical care and management, especially as to diet. Loss of weight and vitality are easier retained than regained. Vegetables and raw fruits must form a large part of the daily dietary. They must be properly sieved to prevent them lodging; lodgment causes esophagitis and increased stenosis. Any food that will go through a mesh of 25 to the inch will pass through a very small lumen, cancerous or tubal. Milk should never be given within three hours of curd-producing foods. Small cancerous fistulæ are more often clogged with curds than with anything else.

Broad and Ontario Streets.

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LXXIII.

III. SURGICAL ASPECTS.*

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ST. LOUIS.

It is unfortunate that this part of the symposium was not assigned to some surgeon who is more optimistic than we are. In spite of occasional dramatic successful operations, the whole story of the surgical treatment of cancer of the esophagus is a most gloomy one.

Apparently the chief difficulties at present with the surgical treatment are not only that in the majority of cases the growth is in a relatively inaccessible portion of the body, but also the fact that most patients who present themselves are late in the respect that they are already suffering from inanition and some degree of cachexia. Early diagnosis seems to be almost impossible now because of a limitation of methods of examination and also because symptoms are not produced, as a rule, until after a considerable involvement of the esophagus has occurred. The most satisfactory methods of examination now in use are the roentgenologic and the esophagoscopic. The latter should be accurate in most cases, but a difficulty which is presented in this method is the inability to recognize with certainty very early lesions. The presence of leukoplakia, which is considered by some as a precancerous lesion, has been shown by Schaer[1] to be perhaps of less significance than ordinarily supposed because in an examination of 200 noncancerous cases at autopsy he found leukoplakia of the esophagus in 60 per cent.

Because of the anatomic difficulties associated with a surgical attack upon this organ it is advisable to consider the condition under headings referable to the particular portion of the esopha-

*Read as part of a symposium at the meeting of the American Bronchoscopic Society, St. Louis, June, 1931.

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TABLE I.

SITE OF CARCINOMA OF THE ESOPHAGUS.

Author—	No. Cases	Upper ⅓	Bifurcation	Lower ⅓
Klein	342	50 (14.6%)	159 (46.7%)	93 (27.2%)
von Hacker.....	...	(12.3%)	(48.8%)	(26 %)
Lotheissen	180	24 (13.3%)	88 (48.8%)	68 (37 %)
Kraus-Ridder ...	1748	469 (27 %)	519 (30 %)	760 (56 %)
Sauerbruch	186	.....	.....	(63 %)

Note the variation in the distribution of carcinoma of the esophagus. Carcinomas at the level of the bifurcation of the trachea are apparently slightly less frequent than is generally stated.

gus that is involved. (Table 1.) The cervical portion is the most accessible to the surgeon, and it is, therefore, not surprising that the operative removal of cancers of this region have been more successful than elsewhere. Even in this location, however, the results are not particularly encouraging. Glück[2] has had the most extensive experience of anybody with cancer of the cervical portion of the esophagus. His operation is a very radical one, which consists in removing not only the esophagus but also the trachea and larynx and the fascia right down to the vertebral column. He has carried out this operation on 114 patients, but despite its radical nature there were only six alive and apparently free from recurrence after five years. His operative mortality was 18 per cent. There are, however, two other patients who have been operated upon by Glück's method who should be mentioned, one operated on by Garre,[3] who was alive twelve and one-half years after the operation, and another one who was living ten years after an operation by Turner.[4] In those cases in which a less radical operation has been performed the results are much less satisfactory. In 1929, Lotheissen[5] collected the records of 82 operations for carcinoma of the esophagus in this region which were less radical than the Glück operation. There was an operative mortality of 22 per cent, but still more discouraging was the fact that only one patient was alive and free from recurrence two years after the operation. Two had survived for one and one-half years, three for one year, and five for more than six months. The next most accessible portion of the esophagus is the abdominal portion. Excluding those cases in which a total gastrectomy was performed for carcinoma of the cardia, there

have been reported five operative successes by the abdominal route (Bircher,[6] Kümmell,[7] Küttner,[8] Völcher[9] and Brun.[10]) However, apparently none of these patients has lived much beyond a year. Zaaijer[11] and Hedblom[12] have each had an operative recovery after a combined abdominal and transpleural operation. The former's case died later of recurrence and the latter's committed suicide.

When we come to the thoracic portion we, of course, encounter the region which has been most baffling from the standpoint of successful surgical attack. The lack of success in dealing with cancer of this region is not due so much to the difficulty of approach to the involved structure as it is to other conditions which exist to make chances of recovery and successful eradication of the growth very slight. The operation of posterior mediastinotomy, particularly the one devised by Lilienthal[13] and performed by him in several of these cases, is not a very difficult or a hazardous procedure. The difficulty of this, as in any other operation for the resection of the involved portion of the esophagus, lies rather in the fact that there is no protecting peritoneal covering over the esophagus to assist in the healing of the anastomosis and consequently there is always great danger of infection of the mediastinum. As a matter of fact, most of the fatalities after these operations are due to a suppurative mediastinitis. One is also confronted by the fact that, even if the involved portion of the esophagus is successfully removed and even if the patient makes an operative recovery, there is still but little chance for him to escape a recurrence of the disease. It is probable that eventually methods could be devised to protect the mediastinum satisfactorily from infection. Several ingenious proposals have already been made. Perhaps one of the most promising of these is the one by Duff Allen,[14] who used a double cuff of fascia lata around the portion of the esophagus to be resected. At the first stage of the operation the esophagus is mobilized and the two layers of fascia are placed around it. At a later stage, after the fascia has healed in place, the outer cuff is incised and the portion to be resected is removed. The gap in the lumen of the esophagus is, therefore, protected by the outer layer of fascia, which has already healed to the surrounding structures. The only really

satisfactory result obtained by the operative removal of a portion of the thoracic esophagus has been the dramatic case of Torek's.[15] This patient lived for thirteen years after her operation and died of pneumonia. At the postmortem examination there was no evidence of a recurrence of the carcinoma. There have been other instances with operative recovery, notably the patient of Lilienthal's, who lived for a year and four months before he died of recurrence. Eggers[16] has also had a successful operative recovery.

No discussion of the surgical treatment of carcinoma of the esophagus is complete without reference to the subject of gastrostomy. There is the greatest possible diversity of opinion concerning the use of gastrostomy. At best this operation is, of course, only palliative, but an important point, which is of interest to all of us, is how much palliation should be expected. Obviously, it becomes imperative to avoid starvation if the patient has a complete obstruction of the esophagus which will not permit the passage of even liquid food. If, however, the patient is able to swallow, the usual experience indicates that gastrostomy not only is not helpful but is positively harmful to the patient. Few individuals yield gracefully to the idea of taking their food and drink for the rest of their lives through a tube into the stomach. Almost always, therefore, following the performance of a gastrostomy a very marked reduction in the morale of the patient and a feeling of hopelessness supervene. At the Barnes Hospital, we prefer in most instances to postpone a gastrostomy until it becomes absolutely necessary in order to avoid starvation. We believe that in the average case it is far preferable to keep the patient swallowing in the normal manner for as long a period as possible, even though it is necessary to undertake some forcible dilatation. In many, perhaps the majority of instances, the lumen of the esophagus can be kept open by the repeated applications of radium to the growth within the esophagus. We believe that it is advisable, even at the risk of doing a fatal injury occasionally to a patient, to keep his normal channel open for as long a time as possible, rather than to perform a gastrostomy unless it is absolutely necessary. Because of the diversity of opinion concerning the usefulness of gastrostomy in cases of carcinoma of the esophagus, one finds very marked differences

in the mortality rates from different clinics. If gastrostomy is postponed until it becomes necessary to avoid starvation, the mortality from the operation will be higher than if it is performed early. At the Barnes Hospital gastrostomy has been performed in 50 cases of carcinoma of the esophagus. (Table 2.)

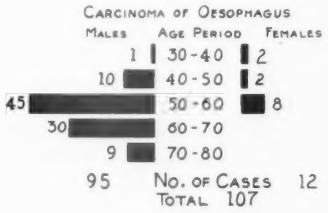

Table 2. Represents 107 patients with carcinoma of the esophagus observed at Barnes Hospital. Almost 25 per cent of these patients were beyond any form of treatment at the time of admission.

In 57 other cases of carcinoma of the esophagus gastrostomy was not performed. Of the series of patients in whom gastrostomy was performed 19 died in less than three weeks, five others died in the succeeding three weeks, one lived for fifteen weeks and two for about eighteen weeks. (Tables 3a, 3b, 4 and Fig. 1.)

TABLE 3A.

CARCINOMA OF ESOPHAGUS.

BARNES HOSPITAL.

Number of cases	107
Dead	31
Gastrostomy	50
Results unknown	19

No.	Year	Age	Sex	Duration of life following Gastrostomy (Dead)	Remarks (Postoperative date patient last observed)
1	1915	52	M	.....	31 days
2	1915	54	M	8 days	Necropsy
3	1915	57	F	4 days	.....
4	1916	50	M	10 days	.....
5	1917	50	M	.....	43 days
6	1917	55	M	4 days	Necropsy

TABLE 3A—Continued.

No.	Year	Age	Sex	Duration of life following Gastrostomy (Dead)	Remarks (Postoperative date patient last observed)
7	1918	59	F	2 months	.....
8	1919	47	F	.....	46 days
9	1920	48	M	.....	32 days
10	1920	54	M	....	44 days
11	1920	65	M	3¾ months	.....
12	1921	71	M	1 day	Necropsy
13	1921	52	M	21 days	.....
14	1922	55	M	29 days	Necropsy
15	1922	59	M	.....	13 days
16	1922	54	M	20 days	Necropsy
17	1922	46	M	7 days	Necropsy
18	1922	53	M	2 days	Necropsy
19	1922	42	M	2 months	.....
20	1922	55	F	17 days	.....
21	1922	55	M	1 month	.....
22	1922	51	M	13 days	.....
23	1923	70	M	17 days	.....
24	1923	52	M	28 days	Necropsy
25	1924	75	M	.....	7 days
26	1924	46	M	4 months	.....
27	1924	50	M	4 days	.....
28	1924	52	M	9 days	.....
29	1924	50	M	.....	6 days
30	1924	60	M	.....	21 days
31	1924	60	M	.....	34 days
32	1924	50	F	.....	42 days
33	1924	62	M	56 days	Necropsy
34	1925	54	M	20 days	.....
35	1925	54	M	2 days	.....
36	1925	61	M	.....	7 days
37	1925	48	M	.....	14 days
38	1926	60	M	20 days	Necropsy
39	1926	58	F	.....	13 weeks
40	1926	72	M	2 days	.....
41	1926	39	F	.....	30 days
42	1926	68	M	.....	21 days
43	1927	59	M	50 plus	.....
44	1927	53	M	16 days	.....
45	1927	57	M	25 days	.....
46	1928	55	M	7 days	Necropsy
47	1929	62	M	.....	17 days
48	1929	62	M	.....	23 days
49	1929	64	M	.....	42 days
50	1929	46	F	4½ months	.....

TABLE 5B.

GASTROSTOMY PLUS ASSOCIATED TREATMENT.

DURATION OF LIFE FOLLOWING GASTROSTOMY.

Number of cases, 50.

Barnes Hospital.

Gastrostomy only (33 cases).
Dead, 23. (1, 2, 4, 4, 4, 6, 7, 8, 9, 10, 13, 20, 20, 20, 21, 28, 29, 56 days; 1 week: 1, 2, 2, 4 months).
Results unknown, 10. (7, 13, 17, 17, 21, 31, 34, 43 days; 6, 13 weeks.)

Gastrostomy plus X-ray therapy. (1 case.)
Results unknown, 1. (42 days.)

Radium plus gastrostomy (12 cases).
Dead, 7. (2, 2, 16, 17, 17 days; 15 weeks; 1 month.)
Results unknown, 5. (7, 32, 44, 46, 50 days.)

X-ray therapy plus gastrostomy (1 case).
Results unknown, 1. (21 days.)

Radium plus gastrostomy plus radium (1 case).
Results unknown 1. (30 days.)

Radium plus gastrostomy plus dilatation (1 case).
Results unknown, 1. (14 days.)

Gastrostomy plus diathermy (1 case).
Dead, 1. (4½ months.)

TABLE 4.

RESULTS OF GASTROSTOMY FOR CARCINOMA OF ESOPHAGUS

Author	No. of Cases Reported	No. of Gastrostomies	Results and Remarks
Miller[1]	29	9	Five patients died a few days after the procedure. Gastrostomy in these cases was apparently performed late.
Mueller and Brill[2]	...	21	Five patients died in less than 90 days; 2 in less than 180 days; 2 in less than 9 months and 3 in less than 12 months. One patient lived over a year. There were 6 deaths; 2 in combination with esophagectomy. Operative mortality 11.8%.
Sherman[3]	50	8	No results given.
Gross[4]	...	167	117 (70%) died in one month and 81% of the patients were dead in 3 months. One patient lived 12 months, another 13 months after gastrostomy.
Wells[5]	...	26	18 patients died in less than 3 months; 5 in the next succeeding 3 months; 3 patients alive at the time report published, 1, 8 and 10 months after operation.
Barnes Hosp. Series	107	50	(Follow ups in 31); 19 died in less than three weeks; 5 others died the succeeding 3 weeks (25 dead in less than 6 weeks); 4 lived for about 9 weeks, 1 for 15 and 2 for about 18 weeks; 28 out of 31 patients survived less than 3 months.

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Perhaps the future will indicate that the greatest promise in the treatment of carcinoma of the esophagus lies in a combination of surgical and radiologic treatment. For the most part, the radiologic treatment has consisted of treatment with the X-ray or by the application of radium to the growth from within the lumen of the esophagus. Probably a more rational procedure would be to use radium also at the periphery of the growth. Because of the resemblance in structure between the esophagus and the mouth, and because of the striking results which have been obtained by the application of radon to carcinomatous growths involving the mouth it seems possible that perhaps equally favorable results might be obtained in some cases of carcinoma of the esophagus if a similar procedure were to be carried out. As a matter of fact, Cleminson,[17] in 1929, seems to have been the first to introduce radon seeds through a thoracotomy wound for insertion into the periphery of the growth in cases of carcinoma of the thoracic portion of the esophagus. Although he tried this procedure in four cases, unfortunately his results are none too encouraging. Perhaps one of the four cases will have a good result, as at the time of the report it was too early to tell definitely. In the other cases the results were not satisfactory. Recently Vilray Blair has treated a patient with cancer of the cervical portion at the Barnes Hospital by the application of gold radon seeds to the periphery of the growth in addition to the intralumenary use of radium, with results which are so satisfactory that they encourage further work in this direction. It would seem to us also as if satisfactory results could be obtained in some cases of carci-

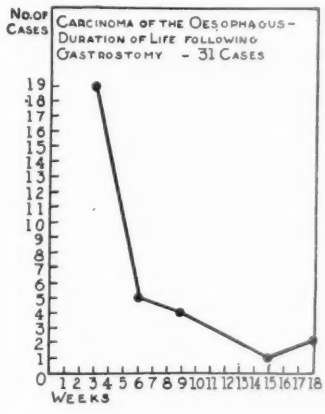

Fig. 1. Fifty of 107 patients with carcinoma of the esophagus were subjected to gastrostomy. The exact duration of life following gastrostomy could only be obtained in 31 cases.

noma of the thoracic portion if the radon seeds were inserted at the periphery of the growth by means of a posterior mediastinotomy. This is an operation which is much better tolerated by the patient than a transpleural approach to the esophagus, such as was used by Cleminson. This procedure would have many advantages over the ordinary methods of attacking the growth by radiotherapy and at the same time it would not subject the patient to any unreasonable risk. Such an operation also would provide much additional information concerning the extent of the growth and the involvement of other structures. By ordinary methods of examination it is very difficult to recognize the presence of metastases unless these are extensive. It is well known, however, that carcinoma of the esophagus is slow to metastasize, as is shown in the accompanying table and figures. (Table 5 and Fig. 2a[18] and 2b.) In the cases in which we have performed posterior mediastinotomy for various reasons, the patient has always tolerated the operation very well, and there has been very little reaction to it. An abstract of Blair's case is appended.

The patient was a female, age 64. For the past year there had been progressive dysphagia and at time of admission to Barnes Hospital on October 23, 1930, she was able to swallow only liquids with difficulty. There had been a loss of twenty-two pounds during the year. A sensation of pain and choking was experienced in the region of the thyroid cartilage. X-ray and esophagoscopic examinations showed the obstruction to be at about the level of the cricoid cartilage. Microscopic examination of the portion of tissue removed through the esophagoscope showed a carcinoma

TABLE 5.

CARCINOMA OF ESOPHAGUS.

STATISTICS OF KLEIN.

No. of cases (necropsy)	264
No. of cases free from metastasis	82
Situation of tumor—14 in upper third of esophagus, 35 at bifurcation, 18, at lower third of esophagus, 4 between upper and middle thirds, 11 between middle and lower thirds.	
No. of cases with involvement of only regionary glands	29

Table 5 indicates the relatively slow growth of carcinoma of the esophagus. Note the high percentage of cases free from metastases and those with involvement of only regionary glands.

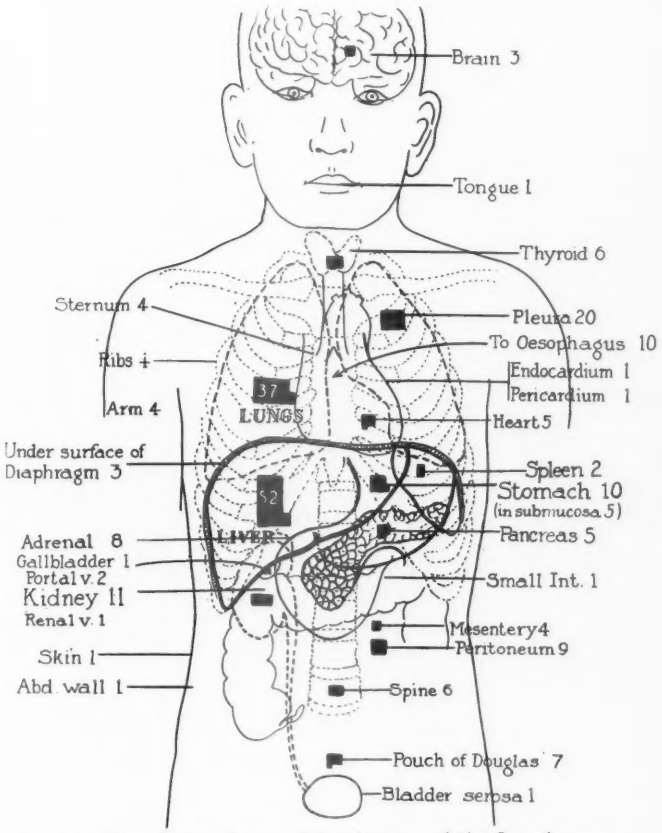

Fig. 2a. The statistics of Klein have been used to illustrate the relative frequency with which the various organs are involved by secondary deposits of carcinoma of the esophagus. The total number of cases concerned in this graphic representation is 264. It is, however, to be noted (Table 5) that in 82 instances there were no evidences of metastases. The liver and lungs were the chief organs involved.

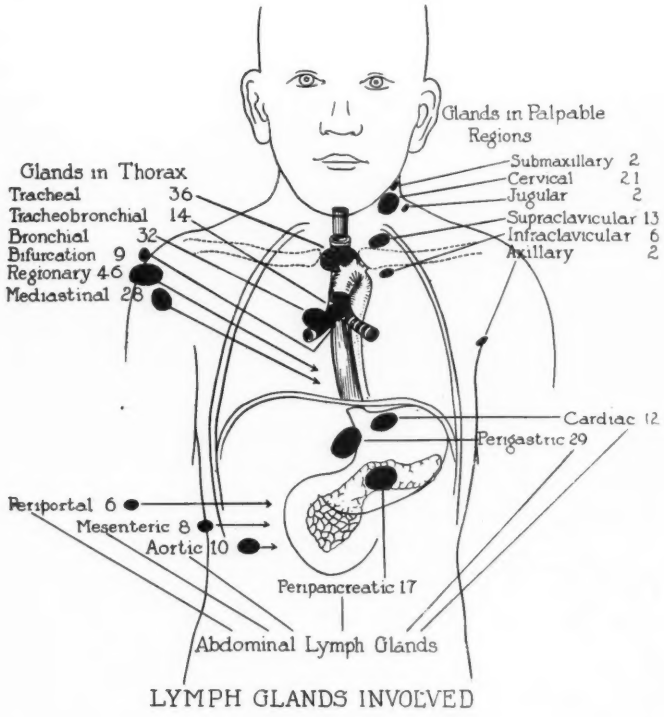

Fig. 2b. (Same as Fig. 2a). This figure clearly illustrates that early extension is by way of the lymphatics and particularly along the vertebral gutter of the posterior mediastinum.

of low malignancy. On November 3, 1930, an incision was made from the angle of the jaw to the articulation of the sternomanubrium junction on both sides. A dissection was then carried into the neck, using care to separate and identify all structures down to the esophagus. Here at about the level of the third or fourth cervical vertebra a mass was found, and 16 gold radon seeds of 1.5 millicurie each were planted outside of this mass, using ten seeds on the left and six on the right. The wound was then closed and rubber drains inserted. The wound healed by pri-

mary intention. A lymph gland which was removed showed no microscopic lesion. Eleven weeks later, January 27, 1931, the patient returned for observation. An X-ray examination at this time showed a very marked improvement in that the former distortion of the lumen of the esophagus had straightened out and a very definite retrogression of the tumor mass was observed. A No. 18 esophageal bougie could then be passed. The patient stated that her swallowing was greatly improved. At this time an attempt was made also to introduce a No. 18 silk bougie containing 150 milligrams of radium down to the strictured area, but the attempt failed. On April 10, 1931, the patient returned for another observation. Great improvement was noticed in the patient's ability to swallow. At this time also a No. 32 bougie could be passed without difficulty. One hundred twenty-five milligrams of radium in a chain of five 25-milligram needles inside a woven silk bougie were introduced into the lumen. There were, however, observed at this time some enlarged glands in the neck, and consequently ten gold radon seeds containing two millicuries each were implanted into these glands.

SUMMARY.

It is possible that carcinoma of the esophagus might be better treated if radon seeds were inserted into the periphery of the growth in addition to the intralumenary use of radium, since both direct surgical attack and radiotherapy alone have been unsatisfactory except for a few dramatic results. The exposure of the tumor, even in the thoracic portion, for the application of radon seeds is not a difficult or a hazardous procedure.

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LXXIV.

TRACHEAL AND ESOPHAGEAL COMPRESSION AS A RESULT OF ADENOMATOUS GOITER.*

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Compression of the esophagus and of the trachea were formerly frequently mentioned in the literature as complications of adenomatous goiter. In earlier literature are numerous instances of patients with adenomatous goiter, who, although apparently in good health, suddenly expired as a result of tracheal compression. One of the tragic experiences of the pioneer surgeons, when operating on the thyroid gland, was that of sudden death from this complication in the course of the operation. Although compression of the trachea and of the esophagus may be produced by other types of disease of the thyroid gland, especially carcinoma, this paper will be limited to compression produced by adenomatous goiter.

With the rapid advance in differential diagnosis of diseases of the thyroid gland, and better understanding of the importance of earlier surgical interference, along with rapid development of surgical technic, large adenomatous goiters, producing obstruction, have become relatively uncommon. In consequence of this improvement, tracheal and esophageal compression have become less common, and proportionally more likely to be overlooked.

Although the large adenomatous goiter is more likely to produce symptoms than the small one, the small, and even the nonpalpable adenomatous goiter may give rise to difficulty. The situation of the adenoma is an important factor in the production of tracheal and esophageal compression. For purposes of convenience, goiters may be listed as cervical, substernal and intrathoracic. Of these, intrathoracic goiter is the most likely to pro-

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*Read before the meeting of the American Bronchoscopic Society, St. Louis, June, 1931.

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duce symptoms of compression, for the region available for expansion is necessarily limited by the bony superior thoracic inlet.

The size and situation of the adenoma are of the greatest significance. However, its shape and its fixation are also contributory factors. This is well emphasized in cases reported by Pemberton, Porter and Spencer, in which the thyroid gland completely encircled the trachea or esophagus. This condition produced very few symptoms in the case reported by Pemberton, but tracheal compression in that reported by Spencer.

As Judd has pointed out, approximately 1 per cent of goiters are entirely intrathoracic. However, dyspnea occurs in approximately a third of all cases of combined intrathoracic and substernal goiters, and dysphagia in approximately 10 per cent. Wuhrmann, however, found dysphagia to be present in only four cases of a series of ninety-one cases of intrathoracic goiter.

Demme, in 1861, first called attention to the alterations in the trachea when it becomes collapsed. Rose, in 1878, gave the first clear conception of the mechanism of tracheal collapse; he noted softening of the tracheal cartilages, although he was unaware of the true pathologic change. He called attention, further, to the influence of respiration on the production of collapse and that a vicious cycle could be established, due to engorgement of the thyroid gland as a result of stridor and dyspnea, with subsequent increase in pressure on the trachea.

That softening of the tracheal rings does not constitute the entire explanation for tracheal collapse has been ably demonstrated by the experimental work of Colp and Louria. They found in dogs that it required removal of at least a third of all cartilaginous rings to produce stridor and obstruction. If the mucous membrane is opened at the same time, however, inspiratory stridor and collapse of the tracheal wall develop. Furthermore, they noted formation of thick, tenacious mucus, and edema of the mucosa, with destruction of the cilia over the compressed portion; these factors are contributory to obstruction. Other factors are the action of the muscles of the neck with change of position of the head, and changes in the annular membrane and mucosal coats.

The treatment for compression of the trachea due to adenomatous goiter is tracheotomy and thyroidectomy. Judd estimated that tracheotomy is necessary in one case of approximately every 500 cases in which thyroidectomy is performed and that, as a rule, restoration of the trachea takes place within a very short time.

The following report is of a case of tracheal collapse due to adenomatous goiter, complicated by the fact that restoration of the lumen of the trachea failed to take place following tracheotomy and thyroidectomy.

Case 1.—A negress, aged twenty-seven years, was first seen at The Mayo Clinic in 1921. At that time her complaint was of an enlarged thyroid gland, dyspnea on exertion, and orthopnea. A diagnosis of multiple adenomas of the thyroid gland was made, and operation was advised. Thyroidectomy was performed, and an enlarged, adenomatous thyroid gland was removed.

The patient did well for four years. Following the birth of a child, she again became aware of gradual, continuing enlargement of the thyroid gland. The only other symptoms noticeable were slightly increased nervousness and dyspnea on exertion. In general, the patient appeared healthy, but the right lobe of the thyroid gland was firm, nodular and fixed. The upper pole of the left lobe was also enlarged, but was less firm, and the trachea was displaced to the left. Examination of the larynx, such as is performed in all cases in which operation on the thyroid gland is contemplated, disclosed that both vocal cords were moving normally. A roentgenogram of the thorax gave evidence of a substernal thyroid gland, with enlargement graded 2 on a basis of 4. The basal metabolic rate was —13 per cent.

A diagnosis of recurring, partially substernal, adenomatous goiter was made. July 1, 1930, the right lobe of the thyroid gland was resected and the isthmus removed. Both lobes were substernal and firmly adherent. The right lobe was estimated to be twelve times the size of a normal lobe. The pathologic report was that there were multitple, hemorrhagic, fibrous, degenerating, colloid and fetal adenomas in a colloid thyroid gland.

Immediately following thyroidectomy a moderate degree of dyspnea and stridor was noted. The vocal cords were moving normally. Stridor and dyspnea became progressively worse, until on the fourth day after operation the patient became unconscious and emergency tracheotomy was necessary. The trachea was found to have been pushed markedly to the left, which made tracheotomy very difficult. Preceding tracheotomy, bilateral abductor paralysis had been discovered. An attempt to plug the tracheotomy tube, or to remove it, a week after operation, produced complete obstruction to respiration. Twenty-five days after operation both vocal cords were still fixed in the median line. In the next three weeks the function of the cords became completely restored. In this period repeated attempts at removal of the tracheotomy tube produced complete obstruction to respiration.

Bronchoscopic examination disclosed the upper limit of complete tracheal obstruction just below the cricoid cartilage. The obstruction was due chiefly to bulging in of the right wall of the trachea and, to a less degree, of the left. The tracheal lumen was easily established by advancing the bronchoscope. The collapse extended to just above the tracheotomy tube. The mucous membrane of the collapsed trachea was edematous but not ulcerated, and markings to suggest tracheal cartilage were not visible.

It was thought advisable to remove the adenomatous goiter on the left side, and to search for a retrotracheal adenoma, which very frequently produced tracheal obstruction. It was also hoped that by causing inflammatory reaction about the collapsed portion sufficient support might be given the trachea to overcome the collapse. At operation the left lobe was found to be retrosternal, and about eight times normal size. Following operation, the patient was able to get air through the larynx, but a few days later the trachea above the tracheotomy opening was again obstructed. Bronchoscopic examination revealed that the trachea was again completely obstructed, but that now, in place of the right wall causing the greatest part of the obstruction, both walls protruded into the lumen of the trachea and met in the median line (Fig. 1).

The patient continues to get along very well with the tracheotomy, but wishes to have another attempt made to restore the tracheal lumen. Whether this can be accomplished is problematic, and is receiving further study.

The question naturally arises whether there is a possible means of anticipating whether a given patient with adenomatous goiter will be afflicted with tracheal collapse. That laryngologic and bronchoscopic examination might be of value was noted by Gibb, in 1863, who reported a questionable bronchocele with bulging into the trachea that was demonstrated by laryngoscopic examination. Although in individual cases definite abnormalities may be disclosed by laryngoscopic examination, such abnormalities will not be revealed in all instances.

Practically the only symptom of value in calling attention to the possibility of tracheal stenosis is stridor. If stridor should afflict a person who had had an enlargement of the neck, but the enlargement had suddenly disappeared, attention should be directed to the possibility of tracheal obstruction from an intrathoracic adenomatous goiter. Hemorrhage into an adenomatous goiter, with tracheal compression, is often described, but it does not as frequently produce tracheal collapse as does displacement of an adenomatous thyroid gland into the thorax.

Hoarseness as a result of paralysis of the vocal cords is frequently mentioned as resulting from an intrathoracic goiter.

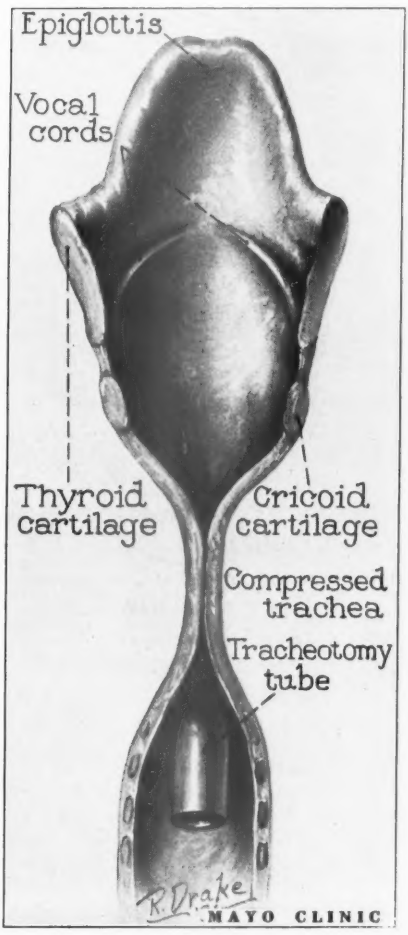

Fig. 1. Collapse of trachea due to adenomatous goiter.

When this occurs, it is more likely to be the result of carcinomatous goiter.

Sgalitzer stated that malacia of the tracheal rings can be distinguished roentgenologically. He wrote that in this condition fluctuation of the tracheal wall can be elicited during respiration by means of the Valsalva and Müller technic, which consists in closure of the glottis and attempt at exhalation or inhalation. In tracheal stenosis without malacia the size of the trachea is reduced, but the lumen is centrally placed, whereas in malacia it is eccentric. Fluoroscopic examination of the trachea will reveal variation in its caliber. However, as Simmonds has demonstrated, ossification and malacia of the tracheal rings may occur in the same patient, and consequently may distort the picture. Fig. 2 illustrates the narrowing of the trachea in case 1, as visualized by roentgenogram.

That dysphagia may also be a complication of adenomatous goiter has already been stated. When it occurs it is usually associated with symptoms of tracheal obstruction. If it is associated with tracheal compression, the etiologic basis is usually readily recognized, but if it occurs without tracheal compression the basic condition may easily be overlooked.

Halpenny, in 1914, reported a case of dysphagia due to compression of the esophagus from adenomatous goiter. His patient, and the one whose case I shall report, received complete relief after thyroidectomy. Interestingly enough, there was no evidence of tracheal obstruction in either Halpenny's case or in mine.

Case 2.—A woman, aged forty years, first came under my observation in 1928. She gave a history of gastric difficulty of long duration for which gastro-enterostomy had been performed and later had to be taken down because of deficient function. She was still having some gastric distress at the time of her visit to The Mayo Clinic. The history relative to the esophagus dated back ten years. The patient first had noted dysphagia when she took solid food, although the same condition might appear when she took liquids. The obstruction was in the "throat," and she had found that she could dislodge the arrested food with a spoon, by a twist of the head, or by massaging the throat. If this did not give relief she would have her physician wash it down by means of a stomach tube. A diagnosis of pharyngoesophageal diverticulum had been made and surgical removal of the diverticulum was advised.

The patient was emaciated, depressed and highly apprehensive, and weighed only 83 pounds. There was a very firm, hard adenoma in each lobe of the thyroid gland. Fluoroscopic examination gave evidence of an

obstruction high in the esophagus, which the roentgenologist interpreted as being due to an extrinsic tumor pressing on the esophagus. Passage of a sound revealed that the obstruction was about 15.5 cm. from the incisor teeth. A 45 French sound, however, could be passed by the obstruction with ease, and the impression was that something was pushed to one side by the sound. There was, however, no improvement in deglutition following this procedure. Esophagoscopic examination, under local anesthesia, revealed an obstruction in the esophagus just below the introitus. The esophagus was displaced to the right, apparently by an extrinsic mass

Fig. 2. Narrowing of the trachea preceding operation.

about 2.5 cm. in diameter. The mass was easily displaced to the left, and the esophageal lumen was reëstablished. Immediately below the level of the mass, a second mass, equal in size to the first, was seen displacing the esophagus to the left. This likewise could easily be displaced. The remainder of the esophagus presented nothing unusual. The lumen of the esophagus therefore followed a sharply angulated letter "S" course in its proximal end. Roentgenograms of the thorax and neck revealed two calcified adenomas which corresponded in every respect to the projections visualized through the esophagoscope (Fig. 3).

A diagnosis of esophageal obstruction due to calcified adenoma in an adenomatous goiter was made. Thyroidectomy was performed and the two calcified adenomas were removed. A thorough examination of the esophagus was made at the same time to be certain that the esophageal obstruction might not be due to some other cause. However, nothing was found. Following the operation, the patient's condition was markedly im-

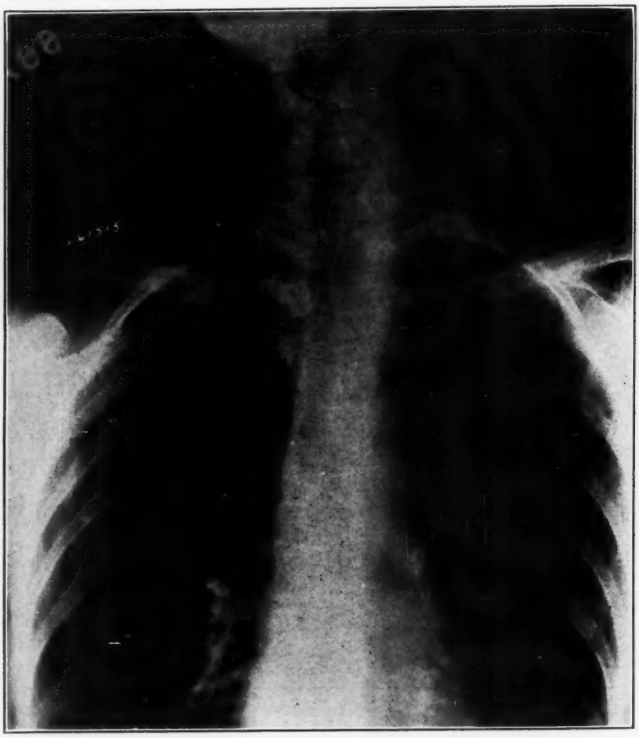

Fig. 3. A calcified adenoma is easily visualized on each side of the esophagus.

proved. She returned again four months later much improved, although a slight amount of dysphagia was still present. Roentgenologic examination gave evidence of nothing more than slight irregularity of the upper end of the esophagus. A 45 French sound could be passed with ease. It was felt that the symptoms still remaining were due to apprehension, for the patient had purposely avoided certain foods because of the fear of strang-

ulation. She again returned for observation two years later and at that time roentgenologic examination of the esophagus was found to be negative.

In conclusion, it may be said that adenoma of the thyroid gland may give rise to tracheal and esophageal symptoms. Such complications have become less frequent due to earlier surgical treatment of disease of the thyroid gland. Moreover, there are normal cyclic variations in the severity of the disease. However, it behooves physicians to be just so much more alert as to the possibility of the occurrence of these complicating factors, because they are now more likely to be overlooked.

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LXXV.

PAPILLOMA OF THE LOWER END OF THE ESOPHAGUS IN A CHILD.*

RICHMOND MCKINNEY, M. D.,

MEMPHIS, TENN.

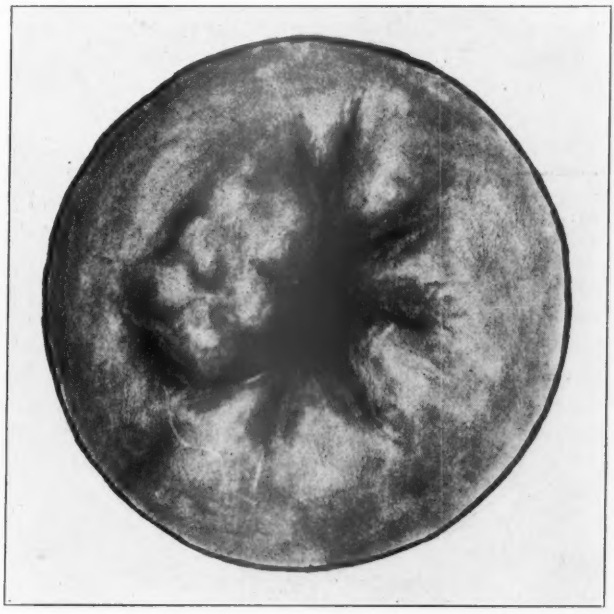
William Spinks, aged three years, Knobel, Ark., was brought to my service at the Baptist Hospital, January 20, 1929, with the history of having several weeks before developed difficulty in deglutition, with regurgitation of solid foods, liquids passing fairly satisfactorily. There seemed to be considerable discomfort in the region of the lower portion of the sternum after food was regurgitated. No history of the ingestion of lye in any form was obtainable. The child was very well nourished. Routine blood examination gave a negative Wassermann.

Fluoroscopy and X-ray pictures showed a constriction at the lower end of the esophagus, just before entrance into the stomach.

My assistant, Dr. C. K. Lewis, who was given the care of the child by me, esophagoscoped him, and at intervals subsequently dilated the stricture with Jackson's linen bougies, and since his ability to swallow food was improved after several dilatations he was returned to his home in Arkansas, with instructions to his parents to bring him back to the hospital from time to time thereafter. This was done, and the child did satisfactorily, but the constriction would close after a short while, and he would be brought back to the hospital for further dilatation.

On April 10th, Dr. Lewis again esophagoscoped the child for examination, and, reading from his notes on the chart, says: "A small esophagoscope was passed to the lower end of the esophagus (hiatus) and at this point the same constriction as formerly observed was noted. The end of the esophagoscope had to be turned sharply to the right to keep the lumen of the esophagus in view. Then a budding tumor, which appeared to come from

*Read before the meeting of the American Bronchoscopic Society, St. Louis, June, 1931.



Drawing made by Dr. C. K. Lewis of tumor as seen through the esophagoscope.

the upper wall of the esophagus, was seen." Examination by me at this time confirmed this finding. A section was taken for biopsy, and a Jackson bougie, No. 26, was passed, but was withdrawn with the greatest difficulty.

The report on the specimen from the hospital laboratory, and also the laboratory of pathology of the University of Tennessee College of Medicine, was that it was papilloma.

On April 12th, under general anesthesia, a large portion of the papilloma, apparently all of it, was removed with a Brünings' cup-shaped cutting forceps, and then a size 22 Jackson bougie was passed several times, this being made possible through removal of the growth. Subsequently a 9 mm. bronchoscope was passed into the stomach.

From this time on the child gained in weight, although a moderate amount of fever followed the operation for several days. On July 7, 1929, the little boy was readmitted, the father stating that three weeks before he had coughed up a mass of tissue of some kind, and since that time his ability to swallow was greatly improved.

Under ether anesthesia, a careful examination was made through the esophagoscope, and no tumor or other abnormality was found in the previously involved portion of the esophagus. Various sizes of Jackson bougies were passed, without the slightest difficulty.

Since then the boy has been brought in at intervals, but no recurrence of the growth has been observed, and while he occasionally has had a slight difficulty in swallowing, it has been found necessary to practice gentle dilatation only, without anesthesia. He is being watched to see if there is any recurrence of the growth, but so far there has been no evidence of its regrowth, and we have not been advised for several months of any return of difficulty in deglutition.

The literature is singularly devoid of reports of cases of papillomata of the esophagus.

Dr. Ellen J. Patterson, before this society in 1927, reported a case of multiple papillomata of the esophagus, occurring in a man, aged 66, and stated that at this time there had been but two cases of papillomata of the esophagus reported, one being

by Chevalier Jackson, in 1907, in a man, aged 36 years, and one by Kernan, of New York, in 1927, in a child, 10 years of age. In Jackson's case, the growth was attached two centimeters below the interarytenoid space, while Kernan's was at the lower end of the esophagus, twenty centimeters from the teeth.

In a personal communication to me, Dr. Jackson says that he has seen three cases that have not yet been published, so I do not know the ages or the location in the esophagus. He says: "It is a rare disease, though probably not as rare as it would be if every human being had an esophagoscopic examination." This statement, of course, might be applied to every other esophageal condition. I believe, however, that we sometimes overlook these cases when we are doing an esophagoscopy, for, as we all know, it is quite easy to override foreign bodies in the esophagus: therefore, why not a soft growth attached to the mucous membrane? I know that in the case that I am reporting this happened several times before the presence of the growth was noted.

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LXXVI.

THE SUPPURATION OF THE PETROUS PYRAMID: PATHOLOGY, SYMPTOMATOLOGY AND SURGICAL TREATMENT.*

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AND

RALPH ALMOUR, M. D.,

NEW YORK.

PART IV.

CASE REPORTS.

In this section we are publishing the reports of nine of our cases of suppuration of the petrous pyramid upon which our observations have been based.

Case 1.—On July 14, 1930, M. K. was admitted to the Beth Israel Hospital, the private patient of Dr. A. A. Schwartz. The child had had an attack of measles five weeks prior to admission and one week later she began to complain of pain in the right ear. A paracentesis of the right drum was performed on June 22, 1930, and an otorrhea had persisted until the date of admission.

On July 14, 1930, blood count showed 3,820,000 red blood cells, with 68 per cent hemoglobin; 15,000 white blood cells, with 76 per cent polys (5 per cent staffs). Otoscopic examination showed a bulging of the postero-inferior portion of the right drum. A central perforation was present, through which protruded several granulations. There were slight mastoid tenderness and periosteal thickening. The temperature, on this date, was 100.6.

The child was kept under observation and was doing well, but on July 20, 1930, she began to complain of occipital headache, and began a slight rise in temperature, to 101.2. A simple

*From the Oto-Laryngological Department of Beth Israel Hospital, New York.

mastoid operation was performed by Dr. Schwartz on that day. The pathology found was that of an acute coalescent mastoiditis. The dura and the sinus were not exposed. The wound was partly sutured and a drain of gauze kept in the lower angle of the wound.

The day following operation the temperature rose to 103.8, and from then on fell by lysis, reaching normal on July 26, 1930. The child was well enough to be discharged from the hospital on July 27th, and appeared to be in good health, the mastoid wound healing satisfactorily.

She returned on July 28th for a change of dressing. On July 29th, the temperature suddenly rose to 103° F., and there then appeared a discharge from the middle ear, which had previously been dry. On July 30, 1930, the child began to complain of severe pains in the right frontal and occipital regions. The temperature was now at a low level, remaining around 101.4; this continued until August 7th, when the child was readmitted to the Beth Israel Hospital and came under our observation.

Examination on that date revealed a moderate discharge from the right middle ear. There was also a slight discharge from the lower angle of the mastoid wound. There were no neurologic signs evident. The child complained of severe temporal and retro-orbital pain on the right side. The blood count on admission was 3,870,000 red blood cells, with 68 per cent hemoglobin; 13,400 white blood cells, with 65 per cent polys; 5 per cent staff cells; 1 per cent eosinophiles. A diagnosis of suppuration of the perous pyramid was made after roentgenographs had been studied and the child was operated upon the following day. An incision was made through the old wound, and granulation tissue, which almost filled the wound cavity, was removed. The simple cavity was then converted into a radical one and a complete toilet of the middle ear undertaken. The overhanging root of the zygoma was next removed and the anterior canal wall considerably thinned with a burr. The middle ear was found to be filled with pus and granulations. The petrosal tip was then entered by means of a burr held at 22 degrees to the external auditory canal and placed one-sixteenth of an inch posterior to the upper margin

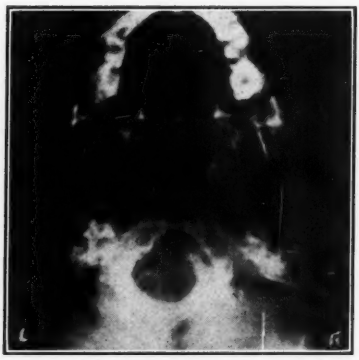

Fig. 19. Case 1. The left pyramid is normally aerated and shows trabeculations. On the right side there is atrophy of the apex and decalcification of the trabeculae, which can still be visualized. The contour of the apex appears slightly irregular.

of the eustachian tube. On entering the petrous tip there was a gush of about a dram of green purulent fluid. The cavity was gently curetted and a rubber dam drain inserted into it. The cavity was then packed with iodoform gauze and the child returned to bed.

The temperature for the next five days varied between 99.8 and 101. The red blood cells and hemoglobin remained at the same level and the white blood cells gradually fell, to reach 9,600 on August 15, 1930, with 55 per cent polys and 2 per cent staffs. The culture of the pus from the petrosal tip showed streptococcus hemolyticus. On August 13th, a full dressing was done, but the drain in the petrosal tip was left in situ. The child's complaint of headache and deep orbital pain had now completely disappeared. From this day on, until August 25th, the temperature remained normal and the child progressed remarkably. All symptoms had by now disappeared and the child was physically in excellent condition.

On August 25th, the drain was removed and a plastic operation was performed upon the external auditory canal and a flap was cut in the posterior membranous wall after the technic of Panse. The posterior wound was closed in and the cavity packed with

rubber dam. Thereafter the wound was handled in the usual manner, to encourage epidermatization of the radical mastoid cavity.

The child was discharged from the hospital on September 2nd, and since that time the radical wound has completely healed and the middle ear is now dry. The child, upon subsequent examination, has been found normal, with a healed radical mastoid cavity on the right side.

SUMMARY.

1. Patient had a well pneumatized mastoid process.
2. Had an attack of acute coalescent mastoiditis for which he underwent simple mastoidectomy with an apparent uneventful recovery.
3. Recurrence of a low grade temperature.
4. Appearance of fifth nerve pain, most intense on right side, and reappearance of middle ear discharge.
5. X-ray showed disease in the right petrosal pyramid.
6. Operation on the petrous tip with recovery.

Case 2.—C. W., a four-year-old girl, was first seen at her home on April 10, 1930. One week before, following tonsillitis, she had developed pneumonia and pain in the left ear. For a while the otalgia disappeared. Then it reappeared and there was a spontaneous rupture of the left drum. For two days prior to our visit there was pain in the right ear. A bulging right drum was found and a paracentesis was performed. The left ear presented an acute purulent otitis media.

The temperature persisted, the ears continued to discharge, and on April 19th we performed a bilateral simple mastoidectomy. An acute coalescent mastoiditis was found on both sides. The mastoids were extensively pneumatized. The mastoid cells were completely broken down and filled with pus and detritus. On both sides the inner table was exposed throughout and the antrum opened. The wounds were sutured with interrupted tension sutures and drained with a rubber dam.

The child made fair progress after operation and appeared to be doing nicely, until May 20th, when a facial weakness was observed on the left side, and the child complained of pain in

the left ear. The right wound was healed at the time and the middle ear was dry. The left middle ear had been dry until May 1st, when a discharge reappeared. The temperature was 100.2. The child was immediately returned to the hospital for observation and study.

On May 21st, the only complaint was of pain in the left ear. Neurologic examination revealed nothing in addition to the

Fig. 20. Case 2. The right petrous pyramid is normally aerated. On the left side there is a slight diminution in aeration but no loss of trabeculations. There is some reactive inflammation at the base of the left pyramid.

peripheral paralysis of the facial nerve on the left side. An X-ray of the mastoids and petrosa was taken and reported the following day. It showed definite cellular development of both petrosæ, with some inflammatory reaction at the base of the left pyramid. Examination of the blood showed 3,690,000 red blood cells, with 59 per cent hemoglobin. There were 14,900 leucocytes, of which 74 per cent were polys (4 per cent staffs). The temperature that evening went to 102.

The following day the facial weakness was more marked and the child continued to complain of pain in the left ear. The temperature ranged between 99.6 and 101.4. The red blood cells numbered 3,740,000, with 59 per cent hemoglobin. The total leucocytosis was 18,400, with 79 per cent polys (6 per cent staffs) and mononuclears 21 per cent. The lower end of the right mastoid began to discharge a slight amount of purulent fluid also.

On May 23rd she was fairly comfortable, although the pain in the left ear persisted. The leucocytosis was 14,600, with 72 per cent polys (9 per cent staffs). The highest temperature was 101.

In the next two days the pain disappeared and by May 25th the facial palsy was gone. The highest temperature on the 24th was 99.4, on the 25th 99.6. A blood count on May 24th revealed 3,540,000 red blood cells, with 59 per cent hemoglobin. The white cells numbered 14,400, with 66 per cent polys (7 per cent staffs). On May 25th she was discharged.

For the next week the child did nicely, making no complaints and running no temperature until, on the evening of June 3rd, when she had developed pain behind the left eye. At this time a diagnosis of suppuration of the petrous tip was definitely made and immediate operation advised but refused.

This was the only complaint until June 9th, when there was a rise of temperature to 103.6 and some stiffness of the neck appeared. A spinal tap then yielded 50 cc. of blood-tinged fluid under 6 mm. of pressure. The next day this was reported positive for albumin (two plus) and globulin, with 1140 cells, 50 per cent polys and 50 per cent lymphs, and a trace of copper reduction. A blood count showed 3,640,000 red blood cells, with 60 per cent hemoglobin. There were 24,800 white blood cells, with 87 per cent polys (5 per cent staffs). Physical examination revealed a moderate degree of cervical rigidity, horizontal nystagmus in both directions, a tendency to a bilateral Kernig and a positive Brudzinski.

Consent for operation was finally obtained, and operation was undertaken that day (June 9th) and the left mastoid wound reopened. After the healthy granulations were removed and the

Fig. 21. Case 2. The right petrous pyramid shows the same appearance as seen in Fig. 7a, taken nineteen days prior to this film. On the left side, however, on comparing with the earlier plate, there are noted a marked diminution in aeration and a distinct loss of the trabeculations in the apical portion. On close inspection, there is a loss in contour of the apex, which was not observed at the original examination.

inner table exposed throughout, the wound was converted into a radical cavity by removing the posterior meatal wall. In the region of the inner wall of the epitympanic space and antrum necrotic, white, friable tissue was removed with the curette, bringing into view the dense bony wall of the superior semicircular canal. The middle ear was curetted and the eustachian tube fully exposed by removing the overhanging zygomatic root. A probe, inserted into the eustachian tube, passed into a fistulous tract extending forward, inward and upward, and toward the petrous tip. There immediately followed a gush of thick, white pus which came out under great pressure along the sides of the probe. A burr was inserted into this area and the opening widened. The cavity was a large one and was thoroughly curetted.

At the termination of the operation there was no facial paralysis. An inequality of the pupils was noted during the anesthesia: the right one was larger than the left. Throughout the operation the anesthetist noticed convulsive movements of both sides of the face which came on prior to the commencement of the surgery.

Following operation the temperature rose to 105.2, dropping later in the day to 103.2. The child slept fairly well that night, the left eye remaining partly open while she was asleep. On the morning of June 10th, the temperature dropped to 101.2. The child complained of severe pain in the left ear and a slight rigidity of the neck was still present. There was pain in the abdomen, which was very hard. The right pupil was larger than the left at one time, although later in the day the pupils were found equal. Opisthotonos was absent. Kernig and Brudzinski were present. The sensorium was clear. Blood count showed a drop in the leucocytosis to 18,600, with 80 per cent polys. That evening the temperature rose to 106.2 and another spinal tap was performed: 50 cc. of cloudy fluid were obtained under 16 mm. of pressure. This was later reported positive (three plus) for globulin and albumin, with a suggestion of a copper reduction and about 600 cells (80 per cent polys, 20 per cent lymphs). A culture from the preceding day's tap showed long-chain streptococci. Fifteen cc. of serum taken from a convalescent erysipelas patient were administered intraspinally.

On June 11th the temperature dropped sharply from 106 to 100.4, rising again later in the day to 102.2. The leucocytosis was again 24,400, with 80 per cent polys (8 per cent staffs). The eyes were swollen and there was an itchy rash over the entire body, for which adrenalin chlorid was given subcutaneously, with subsent disappearance of the rash within several hours. The patient was incontinent and cried with severe pain in the ear and because of headache. The cervical rigidity was more marked than on the preceding day, but not as marked as before operation; some of it seemed due to conscious resistance. A bilateral Kernig was still present; Brudzinski less marked. The pupils were equal and reacted to light and accommodation. Knee jerks were absent; abdominals present. The sensorium was clear.

On June 12th the temperature rose from 101.6 to 104.6. The patient was drowsy, moaning in her sleep, and continued incontinent. She complained of severe headache when she was awake. The leucocytosis increased to 28,000, with 89 per cent polys (6 per cent staffs). The neck was rigid and knee jerks and abdominals were absent. The Kernig was more marked; Brudzinski positive. A spinal tap was done and 35 cc. of cloudy fluid obtained under 16 mm. of pressure. The report on this was albumin three plus, globulin two plus, a trace of copper reduction, 1560 cells with 85 per cent polys. Another intraspinal injection of erysipelas serum (12 cc.) was given, followed in a few hours by a rash on the face which disappeared in three hours.

On June 13th the temperature ranged between 103.4 and 106. The child was very drowsy and did not respond readily. She was incontinent. Blood count revealed 3,510,000 red blood cells, with 60 per cent hemoglobin. There were 22,000 leucocytes, with 84 per cent polys (5 per cent staffs). A cisternal tap yielded 15 cc. of cloudy fluid; and 15 cc. of erysipelas serum were injected. The appearance of the patient was poor; she was cyanosed after the cisternal tap.

The next day the temperature dropped sharply to 99.4, rising subsequently to 106.2. She was pale and her pulse was rather weak; and she had a severe headache which caused her to cry out in her sleep. She was incontinent. Late in the afternoon she failed to respond and tremors of her hands were observed at times. Examination, before the onset of coma, showed some return of the upper abdominal reflexes and the pupils reacted to light and accommodation. Kernig and Brudzinski were present. Some opisthotonos was present. The sensorium was still clear, but the child reacted slowly to questions. A spinal tap was done and 80 cc. of cloudy fluid withdrawn under 50 mm. of pressure. There was again a rise in the leucocytosis to 28,200, with 83 per cent polys (10 per cent staffs).

The temperature was again 106 on June 15th, dropping to 104 and rising again to 106.4. The pulse was weak and there were tremors of both hands. The patient was comatose and incontinent. In her rational moments she complained of pain in the head. A right external rectus paralysis was first noted on this

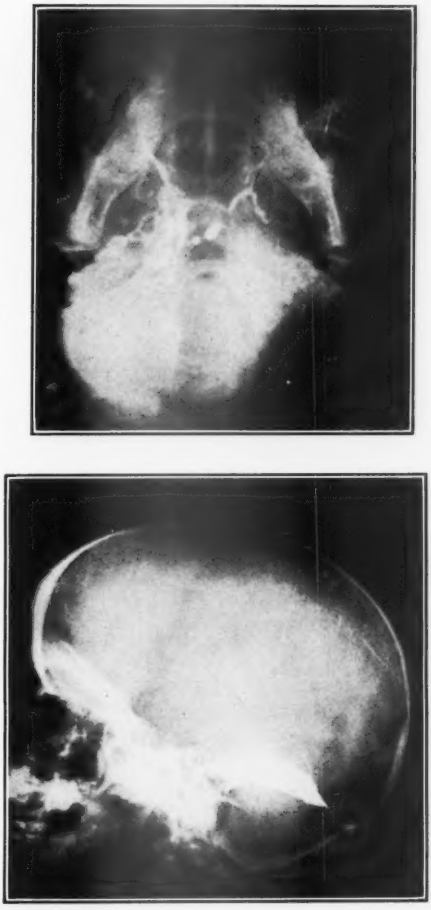

Fig. 22. Case 2. Radiographic examination, postmortem, with lipiodol injected into the fistulous tract. The contrast substance has extravasated into the subdural space and extended posteriorly above the tentorium.

day. A spinal tap yielded 40 cc. of very cloudy fluid under 24 mm. of pressure; and 15 cc. of erysipelas serum were again given. Later that day severe tremors of the entire body set in; Cheyne-Stokes respiration appeared and the patient died.

No postmortem examination was permitted, but an injection of lipiodol was made into the fistulous opening found at operation and an X-ray plate was taken. This showed the lipiodol to have found its way through the petrous tip into the middle cranial fossa, above the tentorium, and over the posterior side of the occipital lobes.

SUMMARY.

1. An extensively pneumatized mastoid process in which a purulent mastoiditis developed.

2. Cessation of the discharge from the middle ear for 19 days after mastoid operation and then the sudden reappearance of the discharge.

3. Forty-five days subsequent to operation an appearance of retro-orbital pain.

4. Transient facial palsy and labyrinthine irritation.

5. Roentgenographic evidence of a lesion in the petrosal pyramid.

6. Operation delayed for six days because of the refusal of the parents and only undertaken when meningitis had already appeared.

7. Fistula found leading into the petrous apex, from which pus exuded under pressure. This finding tends to support Lange's observation that the cells at the mouth of the eustachian tube are the means of egress for pus from the petrous pyramid. The path of invasion in this case was undoubtedly from the epitympanic space.

8. The observation made postmortem that the cranial cavity seemed to be invaded from the petrosal tip to the middle cranial fossa and extending over the occipital lobes on both sides. This may be a factor in the production of the sudden, generalized clinical picture of meningitis, which eventually ensues unless the lesion is relieved while still confined within the anatomic boundaries of the petrosal pyramid.

Case 3.—L. S., a girl of twelve years, was first seen on April 7, 1930. Three weeks before, she had caught cold, and two weeks later she developed pain in the right ear. Paracentesis was followed by a thick yellowish discharge.

At the first examination she was complaining of pain over the right eye, which was swollen. Both physical and radiographic examination revealed mastoid involvement. Mastoidectomy was performed that day.

At operation a mastoiditis of the acute coalescent type was found in an extensively pneumatized process. The usual incision was made, and the cells, which were partly destroyed and contained frank pus, were removed. The mastoid was exenterated to the inner table, exposing the lateral sinus near the knee and lower down toward the posterior meatal wall. The dura was exposed near the zygoma. A large development of the zygoma was found, with infected diploic tissue and cells extending far forward into the root of the zygoma; cellular elements were also found behind the horizontal simicircular canal and downward into the petrous pyramid. These purulent tracts were followed into the depth of the pyramid, and the mastoid tip was also removed. The wound was closed with dermol sutures. A rubber drain was left in situ.

The immediate reaction to operation was entirely usual. On the following day the temperature rose from 99.4 to 103.8, but in the next few days it gradually came down. The child was restless and continued to complain of pain in the ear. Two days after operation she began to complain of a slight abdominal pain, but, although the abdomen was slightly distended, there were no neurologic signs at that time.

On April 8, 1930, the day following operation, differential examination of the blood revealed 3,150,000 red blood cells, with a hemoglobin of 61 per cent. There were 25,500 leucocytes, of which 88 per cent were polymorphonuclears (7 per cent staffs and 81 per cent segmented), 8 per cent were mononuclears and 4 per cent endothelial leucocytes. A slight degree of anisocytosis and poikilocytosis were present.

On April 9th the red blood cells numbered 3,350,000, with 60 per cent hemoglobin. The leucocytes were 26,750 in number, with

88 per cent polymorphonuclears, 8 per cent mononuclears and 4 per cent endothelial leucocytes. Of the polys, however, the staffs had risen to 11 per cent, there were 76 per cent segmented cells and 1 per cent eosinophiles. There was still some anisocytosis. A blood culture taken on this day was subsequently reported negative.

On April 11th, the red blood cells numbered 3,360,000, with a 62 per cent hemoglobin. The white count dropped to 14,750, with 71 per cent polys (11 per cent staffs, 60 per cent segmented, two per cent eosinophiles), 21 per cent mononuclears and 6 per cent endothelial leucocytes. There were a moderate achromia and a slight anisocytosis.

On April 12th the red blood cells were 3,260,000 in number, with a hemoglobin of 60 per cent. The white count continued to drop, to 12,600. There was a rise in the polymorphonuclear count to 76 per cent, of which 12 per cent were staffs, 62 per cent segmented cells and 2 per cent eosinophiles. There were 21 per cent mononuclears and 3 per cent endothelial leucocytes. Achromia, anisocytosis and poikilocytosis were present in a moderate degree.

A culture which had been made of the pus from the ear was reported positive, on April 10th, for staphylococcus albus and a hemolytic streptococcus pyogenes. Throughout this period there was a steadily diminishing quantity of albumin in the urine, intermittent hyaline casts, many epithelial cells and some red and white blood cells.

On April 12th, the fifth day after operation, on which the highest temperature was 101, a full dressing was done and all the stitches removed. There was some stitch abscess; and an edema was present about the posterior scalp edge of the wound. Pus was expressed and the surface painted with iodin and alcohol and cleansed. The middle ear was still discharging; and the child still complained of retro-orbital pain on the right side in occasional spasms.

On April 13th the temperature ranged between 99.6 and 101.6; on the 14th the range was between 99.2 and 101.6. A differential white blood cell count, on the 13th, showed 10,200 leucocytes, with 66 per cent polys (8 per cent staffs, 55 per cent segmented cells, 3 per cent eosinophiles), 29 per cent mononuclears and 5 per cent

endothelial leucocytes. The pain in the eye was less and there was no longer a complaint of cramps.

For the next few days the temperature range was not great—between 99.2 and 100.2 on the 15th, between 99.8 and 100.2 on the 16th, and between 99.2 and 101 on the 17th. Blood examination on the 15th and 16th showed no marked change. On the latter date the retro-orbital pain was almost gone. On dressing, the wound adaptation was found to be satisfactory, and the amount of pus materially less. There was still a considerable middle ear discharge.

On April 17th the patient began to complain of pain in the back of the right leg and of severe pain in the right eye, which appeared more swollen. She was more drowsy. The following day the complaint was of pain in the eye and ear; the patient did not feel well, and there was a sharp rise of temperature from 98.8 to 103. The discharge from both the middle ear and the mastoid was less purulent; and the appearance of the wound was satisfactory. On this day there was a red blood count of 3,265,000, with 60 per cent hemoglobin. The white count rose to 14,750, of which 79 per cent were polys (15 per cent staffs, 64 per cent segmented), 17 per cent mononuclears and 4 per cent endothelial leucocytes. A moderate achromia and slight anisocytosis and poikilocytosis persisted.

The following day, April 19th, the temperature continued septic (from 99.4 to 102.8). The blood picture was about the same, with a very high staff count (16 per cent). A blood culture was taken, which was reported negative after a few days.

A consultation was held, and, in view of the septic type of the temperature and persistent pain in the right eye, re-operation was decided upon. The old wound was laid open, the lateral sinus was examined and was cleared of a thrombus and the internal jugular was ligated.

There was a sharp temperature reaction the following day, from 100 to 104.2. The patient complained of slight frontal headache over the eyes and of pain on swallowing. There was some hoarseness; but the larynx was found to be of normal motility. The leucocyte count rose to 27,900, with 90 per cent polys (17 per cent staffs, 72 per cent segmented cells, 1 per cent eosino-

philes), 7 per cent mononuclears and 3 per cent endothelial leucocytes. She was restless that evening, changing her position frequently and sitting up to do so.

The next day, with a temperature ranging between 101.2 and 102.6, there were 3,390,000 red blood cells; hemoglobin 62 per cent. The leucocytosis was 25,100, with 92 per cent polys (15 per cent staffs, 77 per cent segmented), 5 per cent mononuclears and 3 per cent endothelial leucocytes. The child was very restless and irritable, changing her position frequently. She complained of pain in the ear, difficulty in swallowing and pain over both eyes, at times in the right eye alone.

On April 22nd there was less pain in the ear, but she still complained of pain in the eyes, particularly the right, and there was a recurrence of the pain in the stomach. That afternoon she began to complain of dizziness when turning from side to side. The temperature was between 101 and 101.8 that day; and the blood count was as follows: red blood cells, 3,275,000; hemoglobin, 59 per cent; white blood cells, 15,600, with 68 per cent polys (7 per cent staffs, 59 per cent segmented, 2 per cent eosinophiles), 26 per cent mononuclears and 6 per cent endothelial leucocytes.

On the following day the temperature range was between 100.6 and 102. The patient was still restless and continued to complain of pain in the ear and eyes, chiefly the right, and of dizziness on turning. A slight horizontal nystagmus was observed when the eyes were turned to the extreme right. When the C fork was placed on the forehead, it was heard in the diseased ear. With the noise apparatus in the left ear, she heard the spoken voice in the right. She did not lie in "forced position." A full dressing was done, and all gauze removed except the plugs in the sinus. Then the ear was washed with Dakin's solution and the patient complained of dizziness. A rubber dam was inserted and the neck wound dressed. A blood count on this day showed 3,420,000 red blood cells, with a hemoglobin of 58 per cent; leucocytes, 14,250, with 77 per cent polys (16 per cent staffs, 57 per cent segmented, 4 per cent eosinophiles), 18 per cent mononuclears and 5 per cent endothelial leucocytes.

The following day the child complained a great deal of pain in the right eye; and both eyes appeared slightly swollen, per-

haps due to crying. She continued to complain of pain in the ear and abdominal cramps. She was nauseated at times, but there was no actual complaint of dizziness. The temperature ranged between 99 and 101. The erythrocyte count was 3,560,000, with 60 per cent hemoglobin. There were 12,400 leucocytes, with 83 per cent polys (17 per cent staffs, 63 per cent segmented, 3 per cent eosinophiles), 13 per cent mononuclears and 4 per cent endothelial leucocytes.

On April 25th the temperature ranged between 99 and 101.4, the child continued to complain of a great deal of pain in the right eye and both eyes were swollen. She complained of dizziness in the morning and was drowsy. Blood examination was virtually the same, except for a rise in the hemoglobin to 63 per cent and in the total leucocytosis to 16,150. A full dressing was done and the plugs removed, with no bleeding from the sinus. The stitches were drawn together and tied. The neck was dressed and the wound found granulating.

The following day the child was drowsy. When she was awake, she complained of feeling tired most of the time and was restless. She had severe pain in the right eye; and the eye and cheek were swollen. The temperature was between 99.6 and 101.6. The red blood cells totalled 3,350,000 with a hemoglobin of 63 per cent. There were 19,500 leucocytes, of which 83 per cent were polys (19 per cent staffs, 63 per cent segmented, 1 per cent eosinophiles), 13 per cent mononuclears and 4 per cent endothelial leucocytes.

The next morning, April 27th, there was a sharp rise in temperature, from 100.4 to 104.8. The child continued tired and the pain in the right eye was severe. At times both eyes were complained of. Her memory seemed poor and she began to jerk her leg (the nurse could not determine which, because of her position) and jerkily flex both hands toward her arms; this was observed chiefly on the right side. She was nauseated but did not vomit. There was a spontaneous nystagmus when she shifted position, yet she continued to lateralize sound to the diseased ear, and to hear sounds with the noise apparatus in the healthy ear. A full dressing was done and no pus found either in the neck wound or when washing through the middle ear.

Fig. 23. Case 3. The left petrous bone shows extensive pneumatization and is normally aerated. The left mastoid process is intact. On the right side the operative defect in the mastoid process is seen. From here extending forward and inward the pyramid shows a diminution in aeration with a loss of trabeculations in the apical portion. The apex shows atrophy but no loss in contour.

Neurologic examination was negative except for a slight Kernig and a blurring of the optic disc on the nasal side. X-ray of the right petrosal pyramid revealed a blurring of its outline at the tip. Blood count revealed 3,690,000 red blood cells with 63 per cent hemoglobin. There were 17,850 leucocytes, of which 86 per cent were polymorphonuclear (17 per cent staffs, 68 per cent segmented, 1 per cent eosinophiles), 11 per cent mononuclear and 3 per cent endothelial leucocytes. A spinal tap was done and 5 cc. of clear fluid removed under great pressure. After withdrawal of the specimen, the manometer reading was 26 mm. of mercury. The report received the following day revealed the fluid to be slightly cloudy but sterile; albumin was present, there was a faint trace of globulin; two white cells were found and a moderate number of red.

On April 28th the temperature continued to range high, between 101.8 and 104.4; the pain persisted in both eyes; and there was a nystagmus in both horizontal planes in addition to a slight, vertical, rotary nystagmus; she still heard sounds in the affected ear. Neurologic examination revealed a low grade swelling of the optic discs with blurring of the margins and an increased filling of the veins. There was a right Kernig. The deep reflexes were equal. The left plantar was equivocal. Weber was to the right.

In view of the persistent pain in the eye, the symptoms of intracranial involvement, the septic temperature and the radiographic corroboration, operation was undertaken that day. The packing was removed from the mastoid cavity and the wound opened wide. The sinus was found obliterated and the tegmen above the knee of the sinus was curetted. The mastoid was converted into a radical cavity. There was extensive cellularization of the entire root of the zygoma extending beyond the anterior canal wall and down to the tympanic cavity, which was filled with granulation tissue that was curetted. The malleus and incus were removed and the entire cavity cleaned. An area of necrosis was found superior to and anterior to the horizonal semicircular canal. This extended inward to a depth of ⅛ inch, and forward above the semicircular canal and around it for a depth of ½ inch. It was removed. An opening was made $\frac{1}{16}$

inch posterior to the mouth of the eustachian tube, with a burr directed at an angle of 22 degrees to the external auditory canal inward and forward (as described in Section III of this paper). After trephining for a depth of ⅛ inch, a cavity was entered from which about one-half dram of pus was evacuated, with debris and granulations. This cavity was cleaned out. Its depth, measured from the posterior meatal wall to its termination, was approximately $\frac{9}{16}$ inch. The wound was packed with iodoform gauze and left open.

The patient was very restless after operation and grit her teeth continuously. The pulse was thready but the pain in the eyes was relieved, only to return later in the day, accompanied by some swelling. There was a rise in temperature, from 101.8 to 105.2, and she was drowsy. The fingernails were cyanosed and the face pale. The quality of the pulse improved; but she complained of severe pains in the back and the right leg. She voided in bed. The red blood count was 3,760,000, with 67 per cent hemoglobin. There were 18,800 leucocytes, of which 87 per cent were polys (9 per cent staffs, 78 per cent segmented) and 13 per cent mononuclear.

The next day, April 29th, the highest temperature was 104.4, from which it dropped to 99.2. She still complained of pain in the right eye, of severe abdominal cramps and of nausea, vomiting several times during the day; but she appeared less drowsy and more playful. The nystagmus was less pronounced, disappearing at times. The fundi were practically unchanged, except for some signs of recession in the right disc. The abdominal reflexes were equal. The right knee jerk was diminished and the left absent. Ankle jerks were active. There was the suggestion of a bilateral Kernig, but no rigidity of the neck.

On April 30th the temperature range was low, between 99 and 99.8. A round, red area appeared on the right cheek, but she appeared brighter during the day. In the evening she complained of severe pain in the wound and abdomen and of headache. Both knee jerks were present. The Kernig on the right side was almost gone, although there was still a suggestion of one on the left. The left plantar response was equivocal. The right pupil was smaller than the left; and there was a narrow-

ing of the right palpebral fissure. Ophthalmologic examination showed, in addition, a blurring of the nasal discs with congestion of the blood vessels but no evidences of hemorrhages or exudate.

The next day there was a rise in temperature to 101.6. She still complained of pain in both eyes and, occasionally, in the abdomen; but she was much brighter and more cheerful and appeared improved. The blood count was virtually unchanged, except for a drop in the leucocytosis to 8,400, with 78 per cent polys.

On May 2nd she was very restless and irritable again, complaining of pain in both eyes and the neck, of severe pains and stiffness in the back and of occasional pain in the abdomen. The right eye was more frequently complained of than the left. The temperature stayed between 98.6 and 99.4, however, and she appeared much brighter.

During the next two days the temperature ranged between 99 and 99.8. The child continued to be restless and irritable and there were frequent complaints of pain in the right eye. The abdomen also was complained of occasionally. The leucocyte count was 8,800, with 70 per cent polys (4 per cent staffs, 63 per cent segmented, 3 per cent eosinophiles). The red count was unchanged.

On May 5th there was a sharp rise in temperature to 104.2 and the child was again drowsy and nauseous. She complained of pain in the eyes, especially the right, and of occasional pain in the abdomen. Her neurologic status was unchanged except that the nystagmus to the right was a little more pronounced. A spinal tap was done and 10 cc. of sanguineous fluid were removed, not under pressure. On examination, the blood was shown to be a recent contamination of the spinal fluid, which, three days later, was reported sterile. On this day there was a rise in the leucocytosis to 13,000, with 86 per cent polys; there were 7 per cent staff cells.

The following day the temperature range was lower, between 99.4 and 101.8. The pain in the eyes was still present and that in the abdomen was more severe. The red area which had previously been observed on the right cheek reappeared; and she

complained of a general malaise. The red blood count was 3,690,000, with 62 per cent hemoglobin. There were 10,000 leucocytes, with 77 per cent polys (5 per cent staffs, 71 per cent segmented, 1 per cent eosinophiles).

The abdominal pain and nausea were still complained of on May 7th, as well as the pain in the eyes. The temperature stayed between 99.2 and 100.2. The polymorphonuclear count rose to 86 per cent—7 per cent staff cells—but otherwise the blood picture was unchanged. The next day the pain in the eyes persisted, worse this time in the left; but the abdominal pain was less. The temperature went no higher than 99.8 and the condition of the wounds was good. Her lips, which she had been biting, were swollen and painful.

On May 9th the principal complaint was of pain in the left eye and the left side of the mouth and lips: an ulcerative stomatitis was present. She did not complain of as much pain in the abdomen and appeared brighter. The temperature went to 101. There was no change in the blood picture. The highest temperature on the following day was 100.2; but there was still a complaint of pain in the left eye. Eye examination revealed that the right pupil was still smaller than the left. The fundus picture was unchanged: the left eye was more pronounced than the right.

On May 11th the child was much brighter and more cheerful and the temperature did not go above 100.2. There was a red and sore looking area at the back of the head; but the condition of the mouth and lips was improved. There was still pain in the eye.

For the next ten days the condition of the patient slowly continued to improve. The temperature stayed down and the blood picture was better. The pain in the eyes gradually lessened in frequency; the left was more often complained of than the right. On one day the hip bothered her; on another a red area appeared on the right labia majora. The neurologic picture cleared, however; and there was a definite general improvement.

On May 22nd a plastic operation on the posterior membranous canal wall was done. Granulations were removed from the

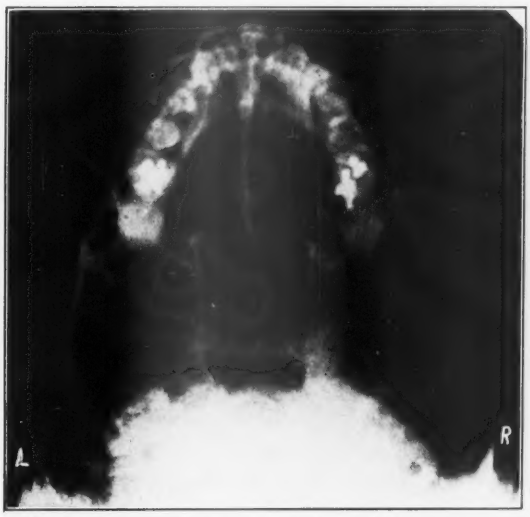

Fig. 24. Case 3. Examination made April 19, 1931, one year post-operatively. The left petrous is normal. The right petrous shows a productive osteitis.

wound and a Panse flap cut in the posterior canal wall and the wound sutured. A rubber dam drain was inserted and a gauze packing.

Progress from this point was rapid; and on June 2nd the patient was discharged from the hospital. Convalescence continued uneventful.

X-ray examination of the petrous bone taken one year post-operatively shows a productive osteitis of the petrous apex.

SUMMARY.

1. Extensively pneumatized mastoid process in which an acute coalescent mastoiditis developed.

2. Constant retro-orbital pain and otorrhea which persisted in spite of adequate drainage from the antrum and middle ear following operation.

3. Development of septic sinus thrombosis, surgical treatment of which failed to cure the patient or relieve the fifth nerve irritation.

4. Transient signs of labyrinthine irritation, spontaneous nystagmus, nausea and vertigo. Hearing present during these periods.

5. X-ray evidence of a lesion in the petrosal pyramid, in addition to the leads toward the tip, eviscerated at the primary operation.

6. Drainage of the petrous tip with evacuation of pus from this area followed by complete cure and healing.

Case 4.—H. S., a girl of thirteen years, was first seen on May 24, 1930. The aural history had begun four weeks before, with a left otitis media purulenta acuta, which ruptured spontaneously. Following this the pain in the ear became extremely severe and the temperature rose to 104. A left mastoidectomy had been done on May 10th by Dr. J. C. Scal, with a temporary subsidence of the pain and the middle ear discharge. Then exquisite pain again set in, on the left side of the head, and the discharge from the middle ear recommenced.

On May 24th the child was obviously acutely ill. The temperature was 104 and she was screaming with pain in the left eye and the ear. A blood count showed 3,500,000 erythrocytes with a hemoglobin of 70 per cent. There were 32,200 leucocytes, with 81 per cent polys (4 per cent staffs, 77 per cent segmented). Radiographic examination of the petrous bone, in which suppuration was suspected, showed evidence of intense atrophy due to decalcification of the septa of the cells of the petrous portion of the temporal bone. There was a localized area of atrophy about the apex of the pyramid. Prior to the operation a spinal puncture yielded cloudy fluid which escaped under moderately increased pressure, and which showed a streptococcus hemolyticus on culture.

Operation was immediately undertaken, the old wound reopened and the granulations completely removed down to the inner table in the mastoid process. The lateral sinus was found exposed and indurated and covered with granulations. It was blocked off above and below and incised between: but no thrombus was found. The hemorrhage was controlled by plugs. A small area of dura in the region of the tegmen celluli was also found exposed. The wound was converted into a radical cavity

Fig. 25. Case 4. The right petrous bone shows pneumatization to the tip and is normally aerated. The trabeculations are clearly defined. On the left side the pneumatic structure is seen to occupy the basal portion of the pyramid and shows a diminution in aeration as compared to the opposite side. The medial or apical portion of the pyramid shows intense atrophy due to decalcification and complete loss of trabeculations. The contour of the apex is intact.

and the middle ear was found filled with granulations and polyps; it contained foul smelling pus. After the toilet of the middle ear was completed, the petrous pyramid was entered by means of a small burr directed at an angle of 22 degrees to the external auditory canal. A cavity was entered from which pus was evacuated under pressure. The procedure was carried still further inward until the approximate site of the apex was reached. The wound was packed with iodoform gauze and a rubber dam and was left open.

The temperature rose to 105 after operation and dropped to 100.8 the following day. There was no facial paralysis but the uvula was drawn slightly to the right. No other neurologic signs were present. The white blood count dropped to 22,000 with 61 per cent polys: there was a rise in the staff count to 11 per cent. All day the child complained of severe pain in the left ear and eye; and she was restless. Toward evening she became nauseous and vomited and began to see double.

On May 26th the left eye was swollen. The child ground her teeth, complaining of severe pain in the back of her neck and generalized headache. An external rectus palsy then appeared on the left side. A nystagmus to the right was observed, together with a rotary nystagmus upwards. The temperature ranged between 101.4 and 103. The red blood count was 3,300,000, with 58 per cent hemoglobin. Leucocytosis 23,600, with 83 per cent polys (4 per cent staffs).

The next day the severe pain in the ear and the back of the head continued. Examination of the eye grounds was negative; and she lateralized the middle C fork to the diseased ear. The blood picture was virtually unchanged. Toward evening she began to complain of pain over the right side of the head too, and at midnight her temperature was 105.8.

On May 28th the lower angle branch of the facial began to show involvement: the nasolabial fold was less pronounced. The upper branches were not involved. Toward evening a moderate rigidity of the cervical spine set in; and a slight Kernig and Brudzinski were present. The temperature, which had dropped to 102 in the morning, rose to 107 at night. The leucocytosis increased to 29,800, with 92 per cent polys. The child was irra-

tional at times and cried almost continually when she was awake, because of the pain in her head and neck. The right ear was red. Respiration was labored.

On the morning of May 29th the child was very restless. She had been irrational most of the night and continued so at times. Her pulse was weak and irregular. The temperature was 104.4 and her general condition was unchanged: there was severe pain in the neck and head. The white blood count rose to 38,200, with 90 per cent polys. A spinal tap yielded 50 cc. of cloudy fluid; and 20 cc. of polyvalent antistreptococcus serum were injected. Respiration was labored and the pulse thready; a red rash appeared over the entire body. The temperature rose to 108 and the patient ceased to breathe.

SUMMARY.

1. Simple mastoidectomy followed, in 12 days, by the onset of retro-orbital pain and the reappearance of a discharge.
2. Purulent meningitis diagnosed prior to drainage of the petrous tip. Streptococcus hemolyticus in the spinal fluid.
3. Radiographic evidence of disease of the apex of the pyramid.
4. Drainage of the petrous tip with evacuation of pus. This was unquestionably the route of invasion into the meninges.
5. Death due to meningitis which was not controlled by the operative procedure.
6. It is of moment to note that although the child had an acute involvement at the second operation, 14 days after the simple mastoidectomy, there were found purulent granulations and polyp formation in the middle ear spaces. This case, too, showed, as part of the symptom complex, transient spontaneous nystagmus with hearing function maintained. The external rectus palsy, facial palsy and deviation of the uvula are taken to be part of the meningeal picture.

Case 5.—L. J., a young man of 22 years, was first seen on October 15, 1929, complaining of a discharge from the right ear which had come on three months previously, following bathing. The evening before his visit to the office he had begun to have pain on the right side of his head. There was difficulty

in nasal respiration. The tonsils had been removed three weeks before.

Physical examination revealed a subacute mastoiditis on the right side, which was corroborated by X-ray. There was tenderness over the region of the right mastoid and the external canal was filled with a white, mucopurulent discharge. Operation was advised.

On October 18th operation was undertaken. On entry into the cortex in the antral region, there was a free flow of purulent material. Necrotic, cellular and eburnized bone were freely intermingled. The lateral sinus wall was covered with granulations which extended for about 1½ cm. anteriorly. The dural plate under the sinus was necrosed. The contents of the mastoid were removed, the antrum exposed and the aditus enlarged. The sinus was exposed until a normal appearing area was reached. The necrotic dural plate was removed, as was the overhang. The wound was washed and a rubber dam drain inserted. Three silk sutures were used for complete closure, except for the drain.

The blood count on the day of operation showed 4,200,000 erythrocytes; hemoglobin 68 per cent. There were 12,900 leucocytes, with 82 per cent polys (8 per cent staffs). The temperature, which had been 99.8 prior to operation, rose to 100.6 after.

The postoperative reaction was very good. The temperature came down to 100 on the day after operation, to 99.6 on October 20th. On that date, although the patient felt extremely well, a moderate nystagmus to the right and a slight nystagmus to the left were observed.

On October 21st the highest temperature was again 99.6 and all the stitches but one were removed. The wound was clean and the patient felt well. Nevertheless the lateral nystagmus to both sides was increased over the previous day. He lateralized tuning fork to the diseased ear.

Although the nystagmus persisted, the patient continued to do very well. The temperature was flat. The facial was intact and there were no signs of intracranial involvement. On October 27th he was discharged from the hospital, with the wound clean and closed and the middle ear dry. Blood count on October 26th

had shown 4,700,000 red blood cells, with 83 per cent hemoglobin. Leucocytosis 9,200, with 74 per cent polys (15 per cent staffs).

After his discharge the patient came to the office for dressing; and, on November 4th he began to complain of a frontal headache, absent during the day, which came on during the late afternoon and lasted all night. He had periods of pain in the right eye, lasting, at times, all day. On November 9th the middle ear recommenced to discharge and on the 11th he was readmitted to the hospital for observation. His temperature on admission was 100.2. There were 4,960,000 red blood cells; hemoglobin 90 per cent. The total leucocytosis was 10,000, with 77 per cent polys (7 per cent staffs). There was a slight tenderness when the lower angle of the mastoid incision was pressed; and the external auditory canal was filled with a thick, purulent fluid. The pupils were equal and reacted to light and accommodation. The facial was intact. There was a bilateral nystagmus present, both horizontal and vertical, when the patient was lying down and when he sat up. He lateralized to the right side. The pharynx was slightly congested.

During the next two days, with the temperature no higher than 99.6, the nystagmus persisted but the headache disappeared. On the 13th the middle ear was dry, but that night there was a slight recurrence of the temperature.

On November 14th, with a temperature between 99.6 and 100.4, there was a horizontal nystagmus to the left and to the right on looking to the left and right. There was none when the eyeballs were in median line. The nystagmus was not associated with vertigo; and there was no vertigo on change of position, laterally or vertically. Hearing was present in the right ear, for both conversation and tuning forks. In view of all this, it seemed that the nystagmus was probably due to an increased intracranial pressure in the posterior fossa, rather than vestibular in origin. Considering the headache, it seemed likely that there was a venous stasis; but, since there were no systemic symptoms and the blood pressure was approximately normal, surgical intervention was deferred. The red blood count on that day was 4,890,000, with 88 per cent hemoglobin. There were

11,600 leucocytes, 75 per cent polys (9 per cent staffs, 65 per cent segmented, 1 per cent eosinophiles) and 25 per cent mononuclears. That night the patient again had a severe nocturnal headache, localized to the right frontal region.

On November 15th the temperature range was again between 99 and 100.4. There was a slight horizontal nystagmus to the left. The blood picture was approximately unchanged.

On the 16th the temperature went to 100.6; on the 17th the highest point was 99.6. Except for the nocturnal headache there was no complaint.

On November 19th an X-ray was taken of the nasal accessory sinuses, revealing a hyperplastic sinusitis only. The pain was still limited to the right supraorbital region. The eyes were of normal mobility. Examination of the fundi was negative. There was a moderate degree of myopic astigmatism found. The temperature, which had been 100.4 the previous day, rose to 102 that evening. The red blood count was 5,050,000; hemoglobin 88 per cent. The leucocytosis had risen to 13,000, with 62 per cent polys (10 per cent staffs, 50 per cent segmented, 1 per cent basophiles and 1 per cent eosinophiles).

On the 20th the highest temperature was 100.8. There was a slight headache but no ocular pain. A moderate discharge was present from the middle ear: the postauricular wound was closed. The patient complained of pain in the neck and painful deglutition. There were an induration and redness of the right posterior pharyngeal wall.

On the 21st the temperature rose to 102, gradually dropping to 99.2 the following day. There were no complaints from the patient. On November 22nd the erythrocytosis was 4,690,000, with 86 per cent hemoglobin. The leucocytosis was 13,400, with 86 per cent polys (1 per cent met., 14 per cent staffs, 70 per cent segmented, 1 per cent eosinophiles). The man continued free from pain on the 23rd, with his highest temperature 100.4.

On November 24th, after three days free from pain or any objective phenomena, the patient presented signs of neurologic involvement. The left pupil was dilated, the right normal, both reacting to light and accommodation. There was a slight horizontal nystagmus to the right. The soft palate and uvula were

pulled to the right, causing a nasal phonation. There was a tendency to flattening and obliteration of the features on the right side but no complete facial paralysis. The patient could not whistle. The frontal muscles were intact. There was no nausea, vertigo, dizziness nor vomiting. Grips were equal. The temperature did not go above 99.2 on that day.

The next morning the patient's condition was practically unchanged, with a temperature range between 98.8 and 100.6. The red count was 4,490,000; hemoglobin 84 per cent. The total leucocytosis was 12,400, with 77 per cent polys. There was a very high staff count—18 per cent. The aural discharge had practically stopped. The left pupil was larger than the right, though this might have been due to an instillation of atropin five days before. Both pupils reacted to light and accommodation, the right more promptly than the left. There was a flattening of the right side of the face with a lessened nasolabial fold. The redness of the pharynx on the right side was diminished and the tongue was deviated to the right. The abdominal reflexes were active. The tendon reflexes of the lower extremities were more accentuated on the left side and there was a tendency to extensor plantar reaction on both sides. Cremacterics were active. There was no Romberg or clonus. All tests, like the finger to nose and finger to finger, were promptly executed, with dexterity and accuracy.

The next day, November 26th, the condition of the patient was unchanged, except that he complained of pain in the right lateral cervical region and there was tenderness along the course of the jugular vein. In view of the persistent pain over the eye and the signs of neurologic involvement, a deep seated abscess in the temporal bone was diagnosed and surgical intervention was considered advisable. Because of the symptoms of 9th and 11th nerve involvement, the site of the lesion was suspected to be near the jugular foramen. The temperature that morning was 100. Blood count showed 4,780,000 red blood cells, with 84 per cent hemoglobin. The leucocytosis was 12,200, with 85 per cent polys (15 per cent staffs).

A postauricular incision was made along the scar of the previous operation. The wound was opened wide and the super-

ficial bony structure curetted. The bony plate over the knee of the sinus was found necrotic and closely adherent to the sinus wall. It was removed. The sinus was thickened but there was no discoloration evidenced. Its lumen was intact. After plugging above and below, the sinus was incised. No thrombus was found. The bulb was isolated but no pathology found here. The wound was not closed.

That night the temperature rose to 104.6 and there was some nausea, which may have been due to the anesthesia. There was no vomiting. The man had some headache, but felt well on the whole. A paralysis of the right vocal cord was noted.

The morning of November 27th the temperature reached 105.6, dropping subsequently during the day to 102.6. The red blood count was 4,670,000; hemoglobin 82 per cent. The leucocytosis rose sharply to 23,800, with 94 per cent polys (19 per cent staffs).

On the following day the temperature again ranged between 101.8 and 105. A slight headache was still present. The pupils were equal and in mid-dilation. There was no nystagmus. The palate still drooped.

On November 29th the temperature range was between 101.6 and 103.8. The man had a generalized headache, but there were no symptoms of intracranial pressure. The droop of the soft palate was still present. There was again a slight drop in the erythrocytosis and hemoglobin; and the leucocytosis rose to 34,400, with 89 per cent polys. The staff count increased to 32 per cent.

On the morning of November 30th the temperature was 103 and there was a radical change in the patient's condition. He was intermittently irrational and twitched occasionally. The right pupil was dilated to the extreme and did not react to light. The facial paralysis was more pronounced: the orbicularis palpebræ did not function. There was a left Babinski; no ankle clonus. The knee jerks on the left side were markedly exaggerated and there was a reduction of power in the left hand.

There was again a very slight drop in the erythrocytosis and hemoglobin. The white blood cells numbered 32,600, with 95 per cent polys (33 per cent staffs). A blood culture taken then was subsequently reported positive for streptococcus mucosus.

A spinal tap was done and 40 cc. of cloudy fluid removed under a pressure of 26 mm. There were 3,000 cells per c. mm., 95 per cent polys. There was no copper reduction. There was a heavy trace of globulin; albumin three plus. On December 6th the fluid was reported positive for gram positive streptococci in long chains, probably the streptococcus mucosus encapsulatus; and the same organism was reported in the mastoid bone.

The patient was comatose and an exploratory operation was undertaken. With a dull probe the region of the lateral lobe of the cerebellum was explored, through the mesial wall of the lateral sinus. Then a circular portion of bone was removed with a drill at the midtemporo-occipital region and an incision made into the dura, but no lesion was found in either place.

The patient died that evening. A postmortem examination revealed an abscess in the petrosal tip which had ruptured into the posterior cranial fossa.

SUMMARY.

1. Simple mastoidectomy for the relief of a subacute coalescent mastoiditis.

2. Transient symptoms of a vestibular irritation; nystagmus and vertigo.

3. Onset of retro-orbital pain and reappearance of a discharge from the middle ear.

4. A progressive involvement of the nerves passing through the temporal bone led to the diagnosis of a deep seated suppuration of the temporal bone.

This case was seen by us before we had devised a technic for entering the petrous tip and, consequently, the operative measures instituted for the relief of this condition did not attack the site of the purulency.

5. Gradual progression with the appearance of a terminal meningitis.

6. At autopsy an abscess of the petrous apex was found which had ruptured into the posterior cranial fossa.

Case 6.—H. F., a boy of eight years, was first seen on February 16, 1930. Five weeks before he had developed a follicular tonsillitis on the left side (tonsillectomy had been performed

two years before). This was followed by a congestion of the ear; and, two weeks before, the left drum had been incised. Four days later, the right ear ruptured spontaneously and began to drain profusely. The temperature was low and there was no pain except some headache and pain in back of the left ear. There was a considerable loss of hearing for conversational purposes.

A subacute mastoiditis on the left side and an acute otitis media purulenta on the right side were diagnosed. X-ray revealed destruction, not complete, in both mastoids. Bilateral simple mastoidectomy was advised.

In the right mastoid, at operation on February 20th, there were signs of beginning chronicity with some purulency in the region of the antrum. The rest of the mastoid cells were diseased and parts were broken down. The sinus plate was necrotic and was removed, exposing normal looking sinus. The mastoid was cleaned down to the inner table throughout. The antrum was opened. The wound was drained with rubber dam and closed with interrupted sutures of B. & B. On the left side, the findings were somewhat similar, except that from the region of the digastric fossa and the peribulbar area there was an escape of a considerable quantity of fluid pus under pressure. The necrosis on this side involved the tegmen cellulae; and this necrotic area was removed. The inner table was exposed throughout and the wound treated in the same manner as the other side. In both ears the lesion was of the coalescent type.

The temperature, which had been 99 on admission, rose to 103 after the operation; and the right eye was greatly inflamed.

On the next day the temperature ranged between 101.8 and 103; and the child was very restless and noisy. He seemed confused and even irrational at times. There was an erythrocytosis of 3,700,000; hemoglobin 75 per cent. There were 17,400 leucocytes; 70 per cent polys.

On February 22nd the temperature ranged between 100.5 and 101.5 and he appeared greatly improved. There were 4,154,000 red blood cells; hemoglobin 70 per cent. The leucocytosis was 2,500, with 86 per cent polys. It was reported that the cultures

taken from the mastoids at operation both showed streptococcus hemolyticus.

The temperature continued to recede and the general condition of the boy to improve. On March 1st he was discharged from the hospital, for ambulatory treatment. The left ear was dry; the right was still discharging. Temperature was normal, 99.5.

On March 2nd the child began to run a low grade temperature, from 101 to 102, at night. In the morning it was usually normal. When this persisted for a few days, he was returned to the hospital on March 5th for observation. Physical examination was entirely negative except for several nodes in the left posterior triangle of the neck. The temperature on admission was 101. Urinalysis was negative. Blood count showed 3,630,000 red blood cells; hemoglobin 66 per cent. There were 11,800 leucocytes, with 74 per cent polys (8 per cent staffs). There was a moderate purulent discharge from the lower angles of both wounds.

For the next week the boy continued to run a temperature ranging from 99 or 99.5 in the morning to 101.5 and 102.5 at night; but he made no complaints and seemed well. The blood count on March 11th showed 4,070,000 erythrocytes; 70 per cent hemoglobin. The leucocytosis was 9,000, with 60 per cent polys (3 per cent staff cells). On March 8th and 10th, the wounds were dressed and found clean. The middle ears were dry.

On March 12th, with the temperature ranging between 99.4 and 101.4, the boy began to complain of pain in his right temple and right eye, toward evening; and this pain recurred almost every evening thereafter. He continued to run a temperature, but no higher than 100.5. On March 17th blood count showed 4,100,000 red blood cells; hemoglobin 70 per cent. There were 7,000 white cells, with 44 per cent polys (2 per cent staffs). On March 18th he was again discharged from the hospital.

The following day the boy returned to the office with a continued complaint of nocturnal pain over the right eye. He was kept under close observation and the mastoids were radiographed on April 7th. The right mastoid showed no pneumatic struc-

ture except for a few small pneumatic cells within the squamosa. No pneumatic structure appeared in the petrous portion. There was diminished aeration in the region of the antrum. The apex of the petrous portion showed decreased bony density and appeared partly destroyed. The sinus wall occupied a median position and was intact. The dural plate was intact. There were no sequestra present. The diminished aeration in the antral region was reported due to a middle ear infection, and the partial destruction of the apical portion of the petrous pyramid probably due to an extension of the infection from the middle ear. The left mastoid showed no pneumatic structure either, except for several small pneumatic cells within the squamosa which were of normal aeration. The antral region was normally aerated. There was pneumatic structure in the petrous portion. The sinus wall occupied a median position and showed a dissolution in continuity, in the sigmoid portion. There were no sequestra present. The dural plate was intact.

The complaint of nocturnal pain over the right eye persisted. There then appeared a slight, but persistent, discharge from the right ear and pain in the ear. On April 10th the child was readmitted to the hospital.

The temperature on admission was 100. Urinalysis was negative. Blood count showed an erythrocytosis of 4,129,000, with a 69 per cent hemoglobin. There were 9,600 leucocytes, with 60 per cent polys (4 per cent staffs). The nasal accessory sinuses were negative, on X-ray, except for diminished aeration of the frontals, the right ethmoid and the right antrum. The cranial cavity showed a slight increase in the convolutional depressions, particularly in the parietal and occipital region, indicating the presence of increased intracranial pressure. The left petrous bone, on radiograph of the mastoids, showed normal aeration. The right petrous bone showed an indistinct but intact apical portion due to diminished aeration.

The temperature on April 11th ranged between normal and 100.4. Toward evening he again began to complain of slight pain in the right ear, which was discharging, and over the right eye. The blood count showed no significant change.

Fig. 26. Case 6. Left petrous bone shows normal aeration. The pneumatization is clearly seen and the trabeculae between air spaces are intact. The apical outline is distinctly visualized. On the right side the apical contour is indistinct but intact and the entire pyramid shows diminished aeration. No trabeculations present.

On April 12th a revision of the operation on the right ear was undertaken. An incision was made anterior to the old scar and the edges retracted. The mastoid cavity was found completely filled with new, dense, connective tissue which covered over the area of exposed sinus. The sinus was again examined and found to be normal. The mastoid was converted into a radical cavity: there was an extensive cellularization of the entire root of the zygoma extending well forward beyond the anterior canal wall and down to the tympanic cavity. The tympanic cavity was filled with sickly granulations; and after the incus and malleus were removed the middle ear was completely cleaned out down to the bony wall all over. An area of necrosis and osteomyelitis was found just superior to the lateral semicircular canal and around it for a depth of ¼ inch. This was completely removed. With a burr, directed at an angle of 22 degrees to the external auditory canal, inward and forward, an opening was

made about $\frac{1}{16}$ inch posterior to the mouth of the Eustachian tube into the petrous tip. After trephining for a depth of $\frac{1}{8}$ inch, a large cavity was entered which contained granulations, pus and debris. These were thoroughly cleaned out. The depth of the cavity, measured from the posterior meatal wall to its termination, was approximately $\frac{9}{16}$ inch. The wound was packed with iodoform gauze and left open.

The temperature, which had been 99.8 prior to operation, dropped to 98 after it. Blood count on that day showed 3,800,000 red blood cells; 66 per cent hemoglobin. Leucocytosis 8,000, with 52 per cent polys (3 per cent staffs). That night he complained of pain in the right ear and abdomen and headache.

The following day, April 13th, the temperature stayed between 100 and 100.4. The boy's general condition was good. The pupils were normal and the abdomen soft. In the late afternoon the pain in the right eye returned, but there were no neurologic signs. There was pain in the right ear.

On April 14th the temperature range was between 99.6 and 100.8. There was no complaint of pain in the eye and the pain in the ear was somewhat lessened. Tuning fork was heard in both ears. With the fork placed in the middle of the forehead, he lateralized to the left. Blood count showed 3,740,000 erythrocytes, with a hemoglobin of 67 per cent. There were 8,400 red blood cells, with 66 per cent polys (3 per cent staffs). On April 15th the temperature rose to 102.4 and the child was irritable all day, complaining frequently of pain in the right ear. The right eye was complained of only occasionally. The erythrocytosis on this day was 4,040,000, with 69 per cent hemoglobin. The leucocytosis was 6,200, with 69 per cent polys (3 per cent staffs).

The temperature range on April 16th was between 99.8 and 101. The red blood count was 3,920,000; hemoglobin 69 per cent. The white cells numbered 7,400, with 54 per cent polys (2 per cent staffs). There was no complaint of pain in either the eye or the ear on that day.

On April 17th the child's condition continued good, with no pain. The temperature ranged between 99 and 100.4. The total erythrocytosis was 3,770,000, with a hemoglobin of 69 per cent.

There were 5,800 leucocytes, 51 per cent polys (2 per cent staffs). An ophthalmologic examination was negative. With the tuning fork on vertex, the patient lateralized to the left; but with the noise apparatus in the left ear he heard in the right.

On April 18th, with a temperature range between 99.2 and 100.6, there was an occasional complaint of pain in the right ear. The blood count on this day showed 4,040,000 red blood cells; hemoglobin 70 per cent. There were 8,000 white cells; 44 per cent polys.

The patient's condition on April 19th was good, with no complaint of pain and a temperature between 99.6 and 100.2. The blood picture showed no change. On the 20th there was some pain in the right eye. The temperature was 98.8 to 100.

On April 21st the boy had a very good day, with no pain whatsoever. The temperature ranged from 98.8 to 100.2. The red blood count was 4,200,000; hemoglobin 72 per cent. The leucocytosis was 9,000, with 43 per cent polys. On the 22nd and the 23rd the improvement continued, except that on the latter date he presented an inflamed area on both arms, like chapped skin. The temperature continued between 98.8 and 102.2. The blood picture was approximately unchanged.

On April 24th and 25th the inflamed areas on the arms gradually disappeared. The temperature range was the same and the child continued to improve, with no recurrence of the pain in the eye or ear.

This favorable state continued, and on April 29th a Jansen flap operation was performed. The old scar was removed and the wound closed tightly except at the inferior angle. A rubber dam drain and gauze packing were introduced through the external meatus and left in situ. The temperature, which was 98.4 prior to the operation, rose to 99.4 thereafter. The red blood count on that day was 4,260,000, with a 73 per cent hemoglobin. The leucocytosis was 8,200, with 64 per cent polys.

On April 30th, with a temperature between 99.4 and 100.4, the child was restless and irritable because of the pain in his ear. He complained of a sore throat, but examination was negative. On May 1st the temperature ranged between 99.4 and 100.8, and the boy continued to complain of pain in the ear.

Fig. 27. Case 6. The left petrous bone is pneumatized and normally aerated. The right petrous bone is pneumatized and shows a productive inflammatory change in the basal portion of the pyramid. The apical portion of the pyramid shows a moderate degree of atrophy with some evidences of trabeculation. The apex is intact.

May 2nd was a comfortable day for him, with no pain and the temperature between 99.8 and 100.6.

The patient continued to improve, with a reduction of the temperature range to normal, 99.8, and a complete absence of pain.

On May 8th the patient was discharged. Both ears are now dry and he is in excellent health.

X-ray of the petrosa taken one year later shows a productive inflammatory reaction in the right pyramid.

SUMMARY.

1. Bilateral simple mastoidectomy on extensively pneumatized mastoid processes.

2. Complete healing of left wound and middle ear.

3. Twenty days postoperatively there appeared retro-orbital pain on the right side, followed the next day by the reappearance of an otorrhea.

4. Diagnosis of suppuration of the petrous apex was made clinically and substantiated by X-ray.

5. Drainage of the petrous tip instituted and pus obtained. Complete recovery of the patient ensued.

Case 7.—P. R., an eight and one-half year old girl, was first seen on May 6, 1929. She was suffering from sore throat and a cervical adenitis. She was seen again two weeks later, on May 20th. Four days before the left ear had begun to pain and the temperature had risen to 102. The day before the ear had begun to discharge. The child had a cardiac murmur.

A left otitis media purulenta acuta with some mastoidal involvement was diagnosed, and the child sent to the hospital for observation. The temperature on admission was 99.6, rising later in the day to 100.4. Urinalysis was negative. Blood count showed 3,950,000 erythrocytes. The leucocytosis was 10,000, with 78 per cent polys (10 per cent staffs).

The child was kept in the hospital for a week, during which time the mastoidal symptoms abated. The temperature was low grade, ranging between 99.4 and 100.5. The blood picture gradually returned to normal: on May 27th there were 9,200 leucocytes with 65 per cent polys (5 per cent staffs); erythrocytosis 3,900,000, with 75 per cent hemoglobin. Upon her discharge from the hospital on that date, the middle ear was still discharging, but all the acute symptoms had subsided.

The child came to the office for treatment, but failed to make progress. On June 7th an increase in the discharge from the ear was noted, and on June 10th a left simple mastoidectomy was advised. The child was admitted to the hospital with a temperature of 99.6. Blood count showed 4,608,000 red blood cells; hemoglobin 80 per cent. The leucocytosis was 13,200, with 70 per cent polys (5 per cent staffs).

At operation a cavity was found in the tip of the mastoid, where free pus was present. There was an acute coalescent mastoiditis. The mastoid process was well pneumatized. A small area of dura, near the zygoma, and a small area of sinus were exposed.

For a few days the child ran a high temperature, to 103.4 on June 11th, to 102.8 on June 12th, and to 102.4 on June 13th. Gradually it came down, however; and, on June 18th, when she was discharged from the hospital, it reached normal. The blood count at that time showed 4,352,000 red blood cells; hemoglobin 80 per cent. There were 11,600 white cells, with 70 per cent polys (7 per cent staffs).

The child came to the office for dressings for the next three and one-half months. During this time the child had transient periods of dizziness, vomiting and nystagmus. On June 30th she first complained of pain through the left eye, which, together with the other symptoms, was transient. This would persist for a day and then suddenly subside. They were coincident with cessation of a discharge from the middle ear and mastoid wound and upon the reassertion of the otorrhea, the symptoms would abate. At one time during this period there was a transient facial palsy which lasted for about three days and then completely cleared up. The secretion which came from the mastoid process was distinctly mucoid in character, while that coming from the middle ear was purulent and foul smelling.

Since it was quite evident that the wound would not heal, and in the presence of good hearing in that ear, a revision of the mastoid operation was undertaken on October 10, 1929. On incision there was a free exudation of pus from beneath the cutis. No bone necrosis nor caries were visible; and there was no pus, nor were there any sinuses in the antral or mastoid region. The regular postauricular mastoidal incision was made and the scar excised. The periosteum was elevated and retracted and the bony structures in the antrum and mastoid region curetted. The wound was washed through and completely closed with five silkworm gut sutures. A drain was inserted in the ear.

Progress even after the revision was very slow. The wound continued to discharge. The child was extremely nervous; and,

on December 7, 1929, after a complete physical examination which was negative, it was decided to separate the child from the mother. She was placed in the Children's Ward and her physical condition improved greatly under Alpine and other systemic treatment. During this period, she ran a low grade temperature for the most part, with occasional rises, never higher than 102.5.

On January 25, 1930, she was again discharged from the hospital and sent south. On March 25, 1930, she returned with a

Fig. 28. Case 7. The petrosae show extensive pneumatization. The right petrous bone is normally aerated. The left petrous bone shows a generalized diminution in aeration. The trabeculations are intact. The apex is intact.

history of a low grade bronchopneumonia. A tuberculous pulmonary lesion was suspected at that time but, after extensive examination, was completely ruled out. The mastoid wound was healed at the time, but there was a persistence of the otorrhea.

On May 19, 1930, X-ray showed extensive pneumatization of the petrosa, without destructive changes. There was a slight diminution in the aeration of the left petrous bone. With this finding we were led to believe that the child had all this time had a deep seated purulent focus within the petrous pyramid

which had been draining out spontaneously through the middle ear. We decided that, in the event the discharge from the middle ear continued, we would eradicate the lesion by draining the petrous tip. However, under conservative treatment, the otorrhea eventually completely subsided and the middle ear, at the time of this writing, has been dry for four months.

SUMMARY.

1. A well pneumatized mastoid process in which an acute coalescent mastoiditis had developed.

2. Despite several operative procedures a persistent otorrhea continued for a period of over a year, during which time transient attacks of retro-orbital pain, vertigo, nystagmus and vomiting presented themselves. At one time there was a transient facial palsy lasting three days.

3. X-ray of the petrosa showed a distinct diminution in the aeration of the pneumatic structure in the left petrous pyramid.

4. Under these circumstances we had diagnosed this case as one of suppuration of the petrous pyramid which has healed itself by spontaneous drainage and which did not necessitate operative intervention.

We believe that had this case been operated upon and the petrous tip drained as soon as the retro-orbital pain appeared, the prolonged course subsequent to our operation would have been materially shortened and the otorrhea would not have been permitted to remain for such a length of time.

Case 8.—J. H. had a tonsillectomy performed on May 17, 1930. One week later she had an epistaxis for which a nasal packing was necessary in order to stop the bleeding. The following day she began to have pain in both ears and two days after this a bilateral paracentesis was performed.

When first seen by us on June 12, 1930, the ears presented a bilateral purulent otitis media. There was some evidence of mastoid involvement on the left side. There was a pulsating discharge from both ears which increased when pressure was exerted over the jugular. The temperature was 104. She was kept under observation until June 20th when the child began

to complain of pain in the frontal and temporal regions; she vomited twice during the day.

A bilateral mastoidectomy was performed on June 21, 1930. Both mastoids were extensively pneumatized and there was a mass necrosis and fluid pus present throughout both mastoid processes. The right lateral sinus was exposed and found normal. The mastoid processes were exenterated down to the inner table throughout and the wounds sutured. The temperature persisted at 103 until June 23rd, when it began to fall by lysis until a level of 99.8 was reached and maintained from the 26th up until the 30th of June. Her blood count, which on June 23rd was 3,400,000 red blood cells, with 59 per cent hemoglobin; 34,200 white blood cells, with 89 per cent polys (7 per cent staffs), gradually fell until, on June 28th, the white blood cells numbered 11,600, with 70 per cent polys (4 per cent staffs).

The child was discharged from the hospital on June 30th and returned to the office for dressings. She was doing well until July 16th, when she began to complain of pain through the right eye and she had a daily temperature between 100 and 101. At this time the diagnosis of suppuration of the right petrous pyramid was made and operation for its relief was advised, but consent was refused, pending further consideration on the part of the parents and the desire for further consultation.

On July 21st and 22nd the pain through the right eye became very severe and the child was apparently drowsy. The temperature was 100.6. At this time the right middle ear, which had previously been dry, showed a profuse foul discharge coming under pressure. A right facial weakness was now noted. On July 24th the temperature suddenly rose sharply to 104 and consent was then obtained for operation.

X-ray examination of the petrous pyramid showed a localized osteoporosis of the tip of the apex of the right petrous pyramid. The blood count was 3,600,000 red blood cells, with 50 per cent hemoglobin; 12,200 white blood cells, with 74 per cent polys (5 per cent staffs). A spinal tap was performed; 10 cc. of clear fluid under ten mm. of pressure were removed and subsequently reported normal.

Fig. 29. Case 8. The left pyramid is normally aerated and shows the trabeculations extending to the tip. On the right side there is a localized osteoporosis of the apex with a loss of trabeculations. The apical contour is intact.

The child was reoperated upon the day of admission to the hospital. After opening the wound and removing granulations and retracting the soft parts, the interior of the mastoid wound was found clean. The cavity was then converted into a radical mastoid wound. In the middle ear purulent granulations were found and were removed. The toilet of the middle ear was completed by removing the ossicles and the bony boundaries of the middle ear were widened by removing the overhanging zygoma and thinning down the anterior canal wall. With a burr directed at an angle of 22 degrees to the external auditory canal, the petrous tip was entered and a large cavity uncovered. As soon as entrance was effected some pus immediately came out. The interior of the cavity was filled with rather thick, soft granulation tissue and it was lightly curetted out. At the area of the solid angle and between the horizontal and vertical semicircular canals, soft, necrotic bone was removed backward for a considerable area. The lateral sinus was examined and found to be normal.

The following day the facial weakness noted was found to have cleared up. Late in the afternoon of July 26th a horizontal nystagmus to the left was noted and bilateral Kernig was present. On July 27th cervical rigidity was noted. On the evening of the 27th the rigidity of the neck had disappeared and there were no evidences of meningeal involvement.

Neurologic examination on July 28th showed the child to be alert and co-operative; pupils small, equal and reacting to light and accommodation; the ocular muscles were normal; no nystagmus; ocular fundi negative; motor and fifth sensory intact; slight facial asymmetry; other cranial nerves negative; no motor weakness but general hypotonia which was probably constitutional; deep reflexes were obtained with difficulty; the right knee jerk was more active than the left; no Babinski or sensory changes; no frank cerebellar signs; no meningitic phenomena. At that time the neurologists did not think that the findings warranted a diagnosis of intercerebral or cerebellar focus. The blood picture on this date had shown a distinct rise in the total number of white cells to 19,200, with 79 per cent polys (6 per cent staffs).

On July 30th the patient became incontinent, was somnolent and the rigidity of the neck reappeared. A spinal tap performed yielded 15 cc. of slightly cloudy fluid under 30 mm. of pressure. Culture from this fluid yielded a streptococcus hemolyticus. As the day went on the left knee jerk could not be elicited. A bilateral Kernig and a right abducens palsy set in. Forty cc. of spinal fluid were withdrawn and 15 cc. of streptococcus scarlatina antitoxin was administered intraspinally. A marked anaphylactic reaction followed immediately upon the administration of this antitoxin, and cyanosis, spasm of muscles and rapid, shallow respirations supervened. Adrenalin was administered intravenously. Later in the day a marked spastic paraplegia of the right side developed with convulsive movements. Chloroform inhalations for the purpose of relieving the convulsions were not successful. The blood count on this date reached 33,200 white blood cells, with 91 per cent polys and 10 per cent staffs. On the 31st of July the patient ceased to breathe. Autopsy was requested but permission refused.

SUMMARY.

1. Bilateral simple mastoidectomy for acute coalescent mastoiditis in well pneumatized mastoid processes.

2. Appearance of right retro-orbital pain and recurrence of otorrhea led to diagnosis of suppuration of the petrous apex. Operation was advised at the time but refused.

3. Eight days after the diagnosis was established permission for operation was obtained. Pus was evacuated from the petrous tip but apparently the local lesion had already spread into the subarachnoid space with the production of a fatal meningitis.

Case 9.—A. B., a boy of nine and one-half years, was first seen on November 18, 1930. In December, 1929, he had had a left mastoidectomy performed. In February, 1930, because of persistent headaches, he had a second operation performed on the left mastoid and a search was then made for an epidural abscess in the middle cranial fossa. In April, 1930, the wound had been reincised. When first seen by us he still had a discharge from the wound but the middle ear was dry. For three weeks prior to his examination, he had been having headaches over the right frontal area, vomiting and pain over the nape of the neck on moving his head. The temperature ranged no higher than 100. At the age of three and one-half years he had had infantile paralysis, which left the muscles of his left foot paralyzed. For one week prior to examination he had had headaches, lack of sleep and general nervous irritability.

Upon examination, a fistula was found leading to the left mastoidal antrum: bare bone was felt. The middle ear was dry. Hearing tests revealed that acoustic function was present: he lateralized to the diseased ear. A radiograph taken prior to operation showed no pneumatization in the left mastoid. There were signs of cortical destruction with destruction of all pneumatic structure. Diminished aeration was visible in the antral area. There was no sequestra. The petrous portion showed fibrotic changes secondary to an infection.

X-ray of the petrous pyramids showed pneumatization in both, most extensive in the lateral portion. There was a marked diminution in the aeration of the left pyramid, with fewer septa

evident in the inner and tip region. Those septa which were visualized were markedly thickened and there was a definite destruction of other septa, indicating an inflammatory lesion of the tip and partial destruction of the apex.

Reoperation was recommended and undertaken the following day. An incision was made through the old scar and through the fistulous tract. Only part of the operative area was found regenerated. The greater portion of the cavity was filled with granulation tissue and muco-pus came from the depths in the area of the zygoma. After the mastoid cavity was cleared of the granulations, it was found that a modified radical operation had been performed the first time, with removal of the outer attic wall. A portion of the squama was also found to have been removed and there was a large exposure of the dura of the middle cranial fossa with brain hernia extending downward into the mastoid wound. A radical operation was then performed and the incus and malleus were removed. Pus was found exuding from the middle ear and from a large fistulous opening present

Fig. 30. Case 9. Shows the probe which has been inserted into the fistulous tract to have entered the cavity in the apex. The apical destruction here is very clearly noticeable when compared to the opposite side.

above the horizontal canal and posterior to the superior canal. Into this fistulous opening a probe could be inserted for a distance of 1½ inches directly inward and slightly forward into the pyramidal tip whose bony trabeculated walls were broken down. The depth of this cavity was bony and bone could be felt all around. It was deemed advisable to defer further surgery until a later date and the wound was packed with rubber dam.

Fig. 31. Case 9. The lipiodol which was injected into the fistulous tract is here seen to be located between the pyramid and the basilar process of the occipital bone, showing that a communication exists between the petrous pyramid and the subdural space.

The day following operation, a radiographic examination was made with a probe in situ; and the tip of the probe was seen to be in a large cell communicating with the petrous tip. Lipiodol, which had been injected, appeared to have escaped from the petrosa into the subdural tissues. The temperature on this day went no higher than 100; and there were no signs of meningitis, although there was a slight left ptosis.

The temperature for the next two days continued to range between 99.2 and 100.6. X-ray taken on November 22nd showed that the lipiodol seemed scattered, but in apparently the same position as previously reported, along the base of the skull in the suture between the basilar process of the occipital bone and the petrous portion of the temporal bone, in the region of the apical portion of the petrous pyramid.

In the next few days there were no changes of any moment. The temperature went no higher than 101.2. The blood picture showed only a secondary anemia. It seemed plain that the lesion was located in the tip of the petrosal pyramid and the basilar process of the occipital bone. Since there was an open fistulous tract leading from the lesion to the middle ear, expectant treatment was continued. There was no evidence of any involvement of the cranial nerves.

On November 27th there was a slight flattening of the left side of the face: the right angle of the mouth was drawn over more rapidly than the left. The third and sixth nerves were normal; and there was no Brudzinski nor Babinski. No rigidity of the neck was present; and other signs of meningitis were absent. The general condition of the patient was better and by November 30th he was symptomless, with the temperature no higher than 99.4.

On December 5th a plastic was performed on the ear. The radical cavity was thoroughly curetted and all granulations were removed. The bony facial ridge was smoothed out with a burr and the annulus tympanicus removed with this instrument. The opening into the petrous pyramid was enlarged by means of a burr. A curved probe passed into it at this time showed the cavity to extend not only inward but forward in a retrolabyrinthine direction. A Stacke flap was cut in the posterior membranous canal walls and the membranous canal further widened by removing a piece of cartilage and throwing a little flap of skin into the wound. The wound was closed with interrupted B. & B. sutures and drained with rubber dam.

The temperature remained low and the boy continued to improve; and on December 27th the postauricular wound was

Fig. 32. Case 9. The right petrous bone is pneumatized and normally aerated. The left petrous bone shows a productive change in the basal portion. The apical portion shows a slight diminution in aeration, with considerable loss of trabeculations. There is a small collection of air in the petrous bone between the base and apex. The apex is partially destroyed. The contrast substance (lipiodol) previously injected, is in the same location as previously observed, viz: between the basilar process of the occipital bone and the petrous apex.

closed. The skin around the fistulous opening was incised and the edges of the fistula were elevated. The posterior skin wall was denuded of epidermis and corium until a raw surface was obtained, sufficiently large to correspond to the under-surface of the anterior flap. Four mattress sutures were taken so as to bring together the edges of the anterior fistulous flap with the newly made skin edge of the posterior wall.

On January 2, 1931, the boy was discharged from the hospital and he is now completely healed, with a dry middle ear.

X-ray of the petrosa six months later shows the lipiodol still present in the area previously noted and, in addition, there is a beginning productive osteitis of the basal portion.

SUMMARY.

1. Simple mastoid operation with two revisions for failure to heal and persistent headaches and pain in the eye and vomiting.

2. Approximately 11 months after the first operation, because of persistent headaches, retro-orbital pain and continued discharge from the mastoid wound, X-ray examination was undertaken and showed a destructive lesion in the petrous pyramid.

3. At operation, the avenue of invasion was found and this was cleaned out, and injected with lipiodol.

4. After the eradication of the focus in the petrous pyramid, all symptoms had disappeared and the mastoid cavity and middle ear have healed completely.

COMMENTS AND RESUMÉ.

In seven of our cases the operation devised by Dr. Ralph Almour was performed for the relief of the condition.* Of these seven, four are now alive. In the instance of case No. 2, operation was not permitted, although insisted upon, until meningitis supervened. In case No. 4, the child already had a purulent meningitis prior to operation and prior to our seeing her. In case 8, the diagnosis was established and operation insisted upon but was delayed because of the desire on the patient's part for further consultation. We feel that in this case, as well as in case No. 2, the outcome would have been quite different had operative therapy been instituted earlier. Case No. 5 was seen by us before we had devised the operative technic described in Section III of this paper. While the diagnosis of a deep-seated suppuration in the petrosa was made, we were not, at that time, able to surgically attack the lesion. Case No. 7 is included in this classification because, from the history, clinical course and radiographic findings the cause of the chronicity is definitely in the cellular

*We have had two more cases of suppuration of the petrous pyramid which are not included in this paper because they came to our attention and were operated upon subsequent to the writing of this article. One recovered and one died of meningitis. They will be reported in detail at another time.

structure of the petrosa. This case has not been operated on for drainage of the petrous tip because there was a sufficient avenue of escape and all symptoms have entirely disappeared. At the time of this writing the ear is completely dry and the lesion seems to have healed itself. Case No. 3 was the one nearest to a meningeal involvement which, after operation, recovered. Here there was clinical data of meningeal import, particularly the eye-ground changes and the Kernig and Brudzinski. From all of the cases it seems apparent that the best results from the surgical therapy are secured when the operation is undertaken before the quiescent phase of the clinical picture develops.

From what happened in all the cases in which we intervened too late, it is reasonable to assume that those which recovered would have had a fatal termination after an acute meningeal flare-up, had we not operated, unless nature, as in the instance of case No. 7, provided an egress for the pus in the petrosal pyramid. To await such an outcome, once diagnosis of the lesion is made with a reasonable degree of certainty, is not to avail oneself of a surgical therapeutic measure which thus far has been able to save half of the cases. We feel certain that once the diagnosis of this lesion is more readily made, and the trend toward inactivity on the part of the surgeon overcome, that cases will be submitted to operation earlier, with larger percentages of recoveries as a result.

CONCLUSIONS.

1. From the foregoing we contend that suppuration of the petrosal pyramid in well pneumatized temporal bones is a lesion which presents a characteristic clinical picture, and is therefore a clinical entity.

2. The operation which we have used, once the diagnosis is established, entails no dangers that a competent otologist cannot surmount once its technic is mastered.

3. While our cases are as yet few, it is our experience at this time that timely surgical intervention is successful in preventing the advent of meningitis. In no case where meningitis was established as existing at the time of operation were we able successfully to avoid a fatal termination.

4. To establish the diagnosis of the lesion in the petrosal pyramid, very detailed and accurate observation of the patient is necessary, and the real significance of transient symptoms must be comprehended, in addition to expert interpretations of the roentgenographs of the petrosal pyramids. In making the estimation of the X-ray films, the diagnostician must differentiate between a breakdown of intracellular structures, osteoporosis, cellularization and presence or absence of the diploic type of petrosal pyramids. The interpretation of the X-ray films thus becomes of very great importance.

51 West Seventy-third St.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Los Angeles, May 27, 1931, during the session of the Pacific Coast Society. Seventy-seven candidates were examined, of which fifteen were conditioned or failed.

An examination was also held in Philadelphia, June 8th, during the meeting of the American Medical Association. Ninety-eight candidates were examined, of which eight were conditioned or failed.

The Board will hold an examination in Indianapolis. September 12th, just preceding the meeting of the American Academy of Ophthalmology and Otolaryngology, which will be held at French Lick, September 14th to 19th. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska, for proper application blanks.

W. P. Wherry, M. D., Secretary-Treasurer.

H. P. Mosher, M. D., President.

Books Received.

Speech and Voice.

By G. Oscar Russell, Ph.D. Director of Phonetics Laboratories and Speech Clinic, Ohio State University. 8vo, 250 pages, cloth, illustrated. New York: The MacMillan Company, 1931. Price, $4.00.

A semi-popular presentation of the author's ideas of sound production in speech and song. Its style is breezy and though a trifle opinionated is interesting and readable. The book is profusely illustrated with roentgenograms of the voice apparatus in an endless variety of sound producing positions. Much of the clarity of these illustrations is sacrificed by combining radiograms with diagrams, often illegibly lettered, so that neither are seen at their best. Altogether a useful manual for teachers of the deaf and for teachers and students of singing.

Clinical Allergy, Particularly Asthma and Hay Fever: Mechanism and Treatment.

By Francis M. Rackemann, M. D. Physician to the Massachusetts General Hospital. Instructor in Medicine, Harvard Medical School, Boston, Mass. 8vo, 617 pages, cloth, illustrated. New York: The MacMillan Company, 1931. Price, $10.50.

Since its beginnings, a quarter of a century ago, the study of allergy has been intensive and extensive, and the number of individual clinical observations has been unbelievably large. Rackemann has co-ordinated these observations and prefaced them with a scholarly dissertation upon their fundamentals—chemistry, biology, immunology.

A casual examination impresses one with the completeness of detail and the comprehensive scope of this book. Closer scrutiny reveals a highly impersonal scientific attitude toward the author's own work and the ideas of others, which is highly commendable in dealing with a subject so controversial as this one is today.

The laryngologist finds himself closely involved in the treatment and diagnosis of allergy and, since many of its phenomena find expression in his field, a certain responsibility devolves upon him of assuming his share in the future scientific unraveling of the problem. He should read this book, not for the chapters on the nose (for he should long since be familiar with their con-

tents) but for the complete and very readable account of fundamental principles as they are now understood. Without this knowledge the daily observation of patients is a superficial mechanical labor; with it, it becomes an absorbing problem—a practical one which cries out for solution, and which bids fair to require the concerted intelligence of the several specialists in the solving.

In its format this volume is well conceived, and the exhaustive material is arranged for easy reference. Technical details, such as the geographic distribution of pollenating plants and a list of all known allergens, are well presented.

The Displacement Method of Sinus Diagnosis and Treatment.

By Arthur W. Proetz, M. D. Assistant Professor of Oto-laryngology. Washington University School of Medicine, St. Louis. A practical guide to the use of radiopaques in the nasal sinuses, with 146 illustrations and a chart. 8vo, 260 pages, cloth. St. Louis: The Annals Publishing Company, 1931. Price, $6.00.

The Syndrome of Gradenigo.

By Alberto Pagano, M. D., Assistant in the Oto-Rhino-Laryngological Clinic of the University of Napoli. Paper. 8vo, 184 pages. Napoli. S. Giovanni Maggiore Pignatelli, 1931.

On the fifth anniversary of the death of Giuseppe Gradenigo, March 15, 1931, the University of Naples dedicated a bust of the famous otologist, and published this very comprehensive review of his work.

Atlas de Anatomia del Organo del Oido.

By Dr. Pedro Belou. Professor titular de Anatomie descriptiva de la Facultad de Ciencias Medicas de Buenos Aires.

This atlas contains a tremendous number of invaluable photographs and drawings and is very comprehensive in its scope; it is to be hoped that it will be translated into English. This book stresses many anatomical features not always well known to otologists, and the relationships are accurately described. It is a valuable addition to the library of any scientific otologist.

Transactions of the Oto-Rhino-Laryngological Clinics of the University of Rome.

Professor Gherardo Ferreri, Director, and Professor Guglielmo Bilancioni. Paper. 8vo, 462 pages, profusely illustrated. Rome: Giuseppe Farri, 1930.

The collected writings of the Clinic of Oto-rhino-laryngology of the University of Rome, for the year 1930, with a dedicatory introduction in memory of Prof. Gherardo Ferreri.

Society Proceedings.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Regular Monthly Meeting, Monday, April 6, 1931.

THE VICE-PRESIDENT, DR. G. A. TORRISON, IN THE CHAIR.

PRESENTATION OF CASES.

Carcinoma of the Esophagus.

DR. T. C. GALLOWAY.

Case 1.—Transpalatal approach for diathermy of carcinoma of the vault of the nasopharynx.

This is an adaptation of an old method of pituitary gland approach in dogs. In this case an epithelioma arising high on the left side of the nasopharynx could not be reached by retracting the palate, so the soft palate was split in the midline, leaving intact the uvula and posterior border. The hard palate was split with a chisel to the left of the vomer. Through the 4 cm. hole provided it was easy to apply the diathermy electrode. Closure was easy and healing by first intention took place except for a 1 cm. area which closed in two weeks. Palate function is now normal.

Case 2.—Surgical diathermy combined with laryngofissure after the St. Clair Thomson method for carcinoma of the larynx.

The very obvious advantage of resection of one ala of the thyroid cartilage with better exposure, greater operative safety and freedom from reaction was emphasized. In one recent case, done with Dr. Cottle, in which the cord had not been fixed, the use of the cutting current for excision and superficial coagulation of the bed provided additional safety. In another case, done four months before, the growth had grown over the aryepiglottic fold and the extrinsic part of the tumor was easily reached with the electrode. Laryngectomy would probably have been no safer and would have been a much more formidable procedure. The

immediate results were good, though, of course, nothing could yet be said of the final outcome.

Case 3. Foreign Body.—A coarse bristle, 4 cm. long, was removed from the right submaxillary duct. A 19-year-old boy was holding this between his teeth when it suddenly disappeared, followed by pain in the floor of the mouth. Three days later the submaxillary gland was swollen and painful, as was the floor of the mouth. An indefinite line of induration was felt along the duct. A No. 2 Bowman probe was passed into the duct, an incision made upon this 1 centimeter back, fine forceps inserted and the bristle extracted from the duct. Symptoms immediately subsided and the incision in the duct healed in one week.

Case 4.—Intubation for carcinoma of the esophagus as a palliative measure.

A tube modeled after that of Souttar, except that it was braced by a longitudinal wire soldered to the rings, was made of coiled German silver wire. The upper coils were expanded to make a flange to hold the tube above the constriction. The tubes were in various lengths from 5 to 7 cm., with an outside diameter of the body of the tube 5 to 6 mm. The carcinomatous esophagus was dilated with Jackson bougies and metal dilators, and the tube introduced by an obturator through a large Bruning tube and left in situ. The first patient, who had been unable to swallow liquids, gained seven pounds in the first week. The second patient, who had had almost complete obstruction, had kept his tube in for four weeks and then it had passed per via natura but left a lumen which permitted swallowing of fine foods. The tube would be reinserted when obstructive symptoms developed. One patient had died with hemorrhage after intubation; one shortly after intubation, of a cardiac death, hardly chargeable to the procedure.

DISCUSSION.

Dr. M. H. Cottle: There is not much to add to what Dr. Galloway has brought out, except that the procedure was undertaken endoscopically, under local anesthesia. Not knowing the amount of reaction that would follow, only a very small portion of each vocal cord was coagulated. Then, about three weeks later,

one-half of the right vocal cord was treated, which gave the patient sufficient breathing space. This was two and a half years ago; she still has enough breathing space and enough voice to continue her work.

Treatment of Carcinoma of the Esophagus.

DR. T. C. GALLOWAY.

(AUTHOR'S ABSTRACT.)

The palliative methods were discussed with presentation of cases of intubation. Surgery has not been satisfactory and there have been only a few cures. A procedure was described which aims at a radical cure of carcinoma of the cervical esophagus. A preliminary pack is placed around the esophagus, low in the neck, for walling off and the development of tissue immunity. This appears to make later manipulations relatively safe, as demonstrated in the dog and in two human cases. Diathermy was used after such a pack in two patients: in one applied through the opened but not isolated organ, in the other after nearly complete separation of the cervical esophagus from its bed. Although the first died after two months from local extension and the second after twenty-one days following a tracheo-esophageal fistula, the experience with them gives some hope for radical cure of this lesion.

DISCUSSION.

DR. J. HOLINGER: Many years ago I drew attention to an early symptom of carcinoma of the esophagus, which later was confirmed by others. The first symptoms are often not from the esophagus but from the larynx. If in older people complaining of hoarseness and cough we find the larynx comparatively normal, we should not fail to investigate the esophagus. I think not enough stress is put on this point, that early cases are overlooked because one suspects extrinsic malignancy in the larynx, whereas it is in the esophagus. Of course, this is an early symptom, long before difficulty in swallowing or even perforation of a well-established tumor direct our attention to the esophagus. If we can discover the condition in the earlier stages I think we can do more for these patients.

DR. EDWIN MCGINNIS: This work requires a lot of courage and I think it should encourage us to do operations on the neck rather than only the mucous membrane.

I have had quite a good deal of experience with carcinoma of the esophagus. In 1919, I examined and treated ten patients with this disease. The longest any of these patients lived was ten months. I have also had some experience with the sensitivity of the esophagus to abrasion, and know how a little external force often starts up trouble in the esophagus and the patient develops a fatal illness. I remember one patient whom I esophagoscoped for diagnosis with local anesthesia, and the discomfort of the procedure made her turn her head to one side. This turning produced a slight injury to the mucous membrane and a fatal illness was brought on by a descending infection.

I also had a case in which a fish bone had punctured the esophageal wall. On examination a small puncture wound could be seen about one-half inch below the introitus. This was the only esophageal injury, but the patient developed a mediastinitis, the infection descending from the puncture wound down the fascial sheaths to the chest. The patient died. The postmortem showed that there was an opening from the mediastinum into the trachea at the bifurcation, where the mediastinum was found to drain into the trachea, and the infection was coughed up by the patient.

Dr. Galloway was wise in using the two-stage operation, because the packing helps to wall off the area. It seems to me that as we gain experience we ought to have some successful cases in the treatment of carcinoma of the upper end of the esophagus.

DR. FRANK J. NOVAK, JR.: I think Dr. Galloway is to be congratulated on his courage in attacking a problem of such gravity as this. The approach to the lesion is one of extreme difficulty and he has, by adapting surgical diathermy to his approach, done something of considerable value. I think the point of greatest importance is the two-stage operation, the preparation of the tissue around the esophagus before attempting any manipulation of the esophagus by diathermy or other measures. It is well to understand that this is not necessarily a matter of blocking and of sterilization. This alone will not protect the mediastinal space,

but the fact that some infection of a mild nature is necessarily brought into the area by the preliminary operation rather prepares the tissue for further manipulation. We know that immediately, within a few minutes, there is immobilization of polymorphonuclears in this area. These are phagocytes. Within a few hours these are reinforced by histocytes, which do their share in protecting the tissue. This has the effect of establishing a local tissue immunity and that, with further surgery, offers some prospect of success. The patient does not go out with this establishment of local tissue immunity, as other patients do.

I am glad Dr. McGinnis is completely sold on surgical diathermy in lesions of the upper respiratory tract. When I presented this matter some eight years ago it was looked upon with considerable skepticism. It was a radical view, and there was some question as to whether the paper should be read. In my experience with diathermy in the larynx, I have found that the preliminary use of radium offers a greater prospect of success than the use of diathermy alone. I think if we can implant radon seeds or radium and allow the patient to rest a while and then diathermize, the chances of relief are greater.

I enjoyed the paper very much and congratulate Dr. Galloway for going into the work and presenting the paper.

DR. T. C. GALLOWAY (closing): I am grateful for the discussion, for I felt somewhat hesitant about presenting material that is still in the period of speculation.

Regarding laryngeal irritation, my impression is that this occurs only when the tumor of the esophagus is large or extends beyond the esophagus. It is my impression that such irritation is a relatively late symptom and usually means extrinsic involvement. If we are to accomplish anything for carcinoma of the esophagus we should get the cases earlier than that.

As Dr. McGinnis pointed out, the esophagus is very susceptible to infection, and that is the reason for such preliminaries. We must remember that these are among the most desperate cases in medicine, and they are a total loss unless we do something. The patients we have had were much happier under the procedures that were used, for they felt a real attempt was being

made to help them. The relief of mental agony compensated them for the discomforts to which we subjected them.

We are indebted to Dr. Novak, for he was the pioneer in Chicago in diathermic surgery in our specialty, and certainly from his work, which probably had no better prospects at the outset than this, much good has come to all of us. I am glad he pointed out the importance of local immunity in these cases, for I think that is probably a larger factor than the mechanical blocking off of the infection.

I think perhaps the three-stage operation, in which at the second operation only the esophagus is opened and the wound flooded with secretion, will prove better than the two-stage procedure.

After intubation our patients have had no difficulty in swallowing purees and have eaten finely divided meat and vegetables.

The Development of Head Lights and the Introduction of the Good-Lite.

DR. PALMER GOOD.

(ABSTRACT.)

Before describing the various kinds of head lights, the writer deems it advisable to list the qualities of a head light which would make it ideal for the otolaryngologist.

1. It should be useful in every kind of examination or operation, including tonsillectomy, nasal operations, radical mastoids and bronchoscopy.

2. It should produce a brilliant field of illumination of about 1,000 lumens, or foot candles. It must be brilliant, because many of the cavities are illuminated obliquely, and a relatively small amount of light is reflected.

3. The field of light should be uniform, the vertical and horizontal diameters about equal, and adjustable from three-fourths inch to three inches in diameter.

4. The rays should be parallel so that no sharp focal point exists.

5. Co-axial illumination should be possible.

6. The source of light should be adjustable so that it can be worn between the eyes, over one eye, or at the side of the head.

7. The beam of light should be adjustable so that it can be deflected sharply down for general tonsillectomy or examination of patients in bed.

8. The aperture of the mirror should not be smaller than the average size of the pupil of the eye, so that the amount of reflected light returning to the eye is not reduced. This is about one-fourth inch.

9. There should be no glare from the edge of the aperture.

10. Standard bulbs should be used, as they are readily obtainable and low in cost.

11. There should be no possibility of shock.

12. Overheating must be avoided.

13. Durability must be incorporated so that the light will have a long life, be reliable, and enable the owner to carry it with him in his bag.

14. A single adjustment should suffice for an entire operation.

Because of the fact that no head light was available that fulfilled even a majority of the requirements stated, the writer began the construction of a head light about ten years ago. Since that time five models have been made, but the latest one is now presented to the profession for the first time, confident that it fulfills all the requirements.

It is superior to the groups using only an electric bulb or an electric bulb in connection with a condensing lens, because it is much brighter, does not overheat, and because it can be worn close to the head, between the eyes, it makes the wearer unconscious of the source of light and allows a shadow free stereoscopic view of the mouth, larynx or plane surfaces. It is superior to the reflecting type of head light because there is no sharp focal point and it is much brighter.

In bronchoscopy and esophagoscopy the "Good-Lite" is used with the endoscopic tubes of Killian, Bruening or Haslinger. The advantages over other methods are many, but particularly as follows: more light, no electrical connections to the tubes, no

interference with instrumentation, protection to the operator's eyes or glasses and illumination that penetrates far beyond the end of the tube.

DISCUSSION.

Dr. Edwin McGinnis: At the meeting of the Eastern Section of the American Triological Society, several years ago, there was some discussion among the men regarding the different types of lights, and Dr. Yankauer said that the Kirstein light which they were using gave very much better illumination than the distal lighted tubes.

I have found, in my work, that the distal light loses its effect if much secretion and blood are present. I think that Dr. Good has something, and I hope his new bakelite arrangement will reduce the heat of the light, for one of his first lights became very hot after it was burning for a time.

Dr. Robert H. Good: I had been a user of the Kirstein light from the beginning, until about ten years ago, when my son made a light for me. I find as I get older I need more illumination. I had difficulty in seeing for tear sac operations with the Kirstein light, but with the Good-Lite I have little difficulty. Since using the Good-Lite I have discontinued the Jackson tubes. On several occasions with Jackson tubes I went into the trachea and bronchus and failed to see the bifurcation, which is impossible with the Good-Lite. After using the Jackson outfit for three years I went back to a good bright light. On many occasions when you enter the trachea you can see the foreign body in the bronchus. The Haslinger tube is larger and there is more working space for the forceps. It is a great advantage to have a strong light which illuminates far beyond the end of the tube. In the three years I used the Jackson outfit I failed to remove a safety pin and a dental burr. The Gasserian ganglion operation can be done with half the usual opening by means of this light.

Dr. Palmer Good (closing): The light, of course, does become warm, but it will not burn the operator's head or hand. I appreciate the remarks of Dr. McGinnis particularly, because, although he is a user of the distal lighted tubes, he appreciates the advantages of proximal lighted tubes.

DR. J. HOLINGER presented roentgenograms and reported a case of

Cyst in the Right Maxillary Cavity.

The patient was a man, aged 39, who while under treatment for an infection of the nose developed an iritis of the left eye. He was examined by Dr. Harry Gradle, who found an iritis. Roentgenograms showed the infection on the right side and one plate revealed a round shadow in the right antrum. The antrum thereupon was filled with lipiodol, and another roentgenogram showed the shadow very clearly. (Demonstration of X-ray film.) At a Denker operation portions of the polyp or cyst were removed.

The patient made a rapid recovery and the iritis promptly disappeared.

DISCUSSION.

DR. WALTER H. THEOBALD: This case illustrates definitely something I have mentioned before, in that just where you expect the greatest opacity there is a little light passing through the lipiodol, although it does not sharply mark out the area. In the center of the area in the lateral view there is a little light in this locality. In the antral view this does not show, and I should say the growth must have been very anterior. In the lateral view we find the clue to the condition.

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